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# JOURNAL

OF THE

# Indian Medical Association

VOLUME XXI
NUMBER 2

CALCUTTA
NOVEMBER 1951

Exchange Copy.

**Office of Association Hd. Qrs.**
Hanging Bridge, Faiz Bazar,
Daryaganj, Delhi.

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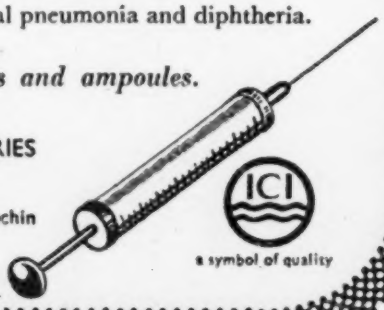

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# APPEAL TO THE MEMBERS

## 1. I. M. A. BUILDING FUND.

The Indian Medical Association has decided to construct a building for housing the I. M. A. at its head-quarters at Delhi. The Association needs about five lakhs of rupees for this purpose. This desire of ours can hardly be fulfilled unless the members of the Association individually come forward to subscribe to the funds which are being raised specially for this purpose.

May I take this opportunity to appeal to each member of our Association to contribute liberally to this fund. The scheme might be realised if every member undertakes to contribute at least Rs. 50/- at a time or in instalments of Rs. 5/- per month.

Those members who have not yet contributed to this fund are requested to please send their contributions for this purpose by crossed cheques or postal orders drawn in favour of "Indian Medical Association–Building Fund" to the Hony. General Secretary, Indian Medical Association, Hanging Bridge, Daryaganj, Delhi 7, at their earliest convenience.

## 2. "YOUR HEALTH".

A health journal meant for the lay people by the name of "YOUR HEALTH" will be published monthly by the Indian Medical Association from January 1952. The price of a single copy will be twelve annas and the annual subscription Rupees Eight only. It is hoped that every member and subscriber will subscribe to this journal.

Members and subscribers are requested to kindly fill up the order form overleaf and send it to the Journal office before the 30th November 1951.

PATNA.
25. 10. 51.

**T. N. Banerjee,**
**President,**
**Indian Medical Association**



(P. T. O.)

# ORDER FORM

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IN BLOCK LETTERS

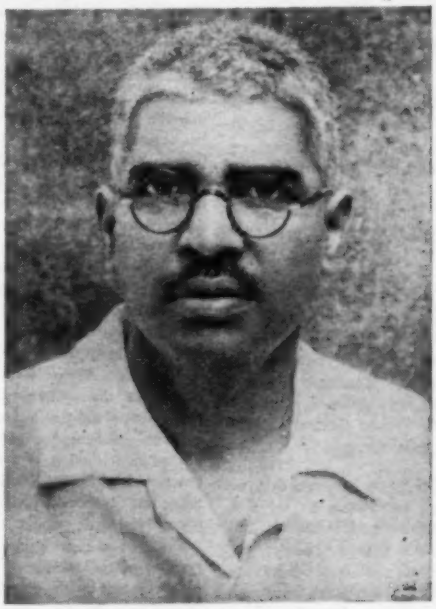
SUPPLEMENT
Journal of the Indian Medical Association

VOL. XXI, No. 2 CALCUTTA NOVEMBER, 1951

**XVII ANDHRA PROVINCIAL MEDICAL CONFERENCE**—The seventeenth Andhra Provincial Conference was held at Rajahmundry on 4th and 5th March, 1951, Dr. G. V. Hanumantha Rao of Guntur presiding.

The Chairman of the Reception Committee, Dr. R. Jayaramaswami Naidu welcoming the audience said that no nation-building activity sould be tackled effectively giving top priority to problems relating to public health and medical relief. It was an admitted fact, said Dr. Naidu, that diseases were rampant in the country and medical relief scarcely touched the fringe of the population. In order to supply medical men for rural areas they had to turn out more medical graduates and should have a larger number of colleges with the specific condition that the graduates should be willing to put in a certain number of years' service in rural areas. Dr. Naidu concluded his speech with the observation that effective methods of treatment of leprosy and tuberculosis had to be thought of and that, eventually, the responsibility of providing medical relief was that of the Government.

Dr. T. Lakshminarayan, Director of Public Health, inaugurated the Conference with a short speech on Public Health. The 'Dr. P. Gurumurti Memorial Prize Essay Gold Medal' was then awarded to Dr. K. Somayya, of Kakinada for the essay on the 'role of a General Practitioner in the prevention of diseases'.



DR. G. V. HANUMANTHA RAO,
*President, XVII Andhra Provincial Medical Conference, Rajahmundry*

The Scientific Session presided over by Dr. M. V. Radha Krishna Rao of Bombay was notable for its symposium. Dr. V. R. Khanolkar of Bombay spoke on the subject of cancer and the effective researches conducted at the Cancer Institute, Bombay. The Medical Exhibition was opened by Dr. Chaganti of Vijayada.

The next year's conference was invited to Rayalaseema.

## PRESIDENTIAL ADDRESS

As we meet to-day in this great and historic town of Andhras I recall to memory the dynamic personality of the late Dr. P. Gurumurti. The organisation of the Andhra Provincial Branch of the I.M.A. was in a large measure due to his relentless work. His multifaceted activities in public life are well-known and it is for us to emulate his selfless humanitarian work. His body perished but his spirit lives to guide and bless us in our endeavours.

Independent India is now three and half years old. Viewing in retrospect this young Republic has not been able to do much either in the matter of promoting health or enriching our country. Truly, we cannot expect a miracle in this short span, but it gives me pain that all things are not going in the right way and there does not seem to be much brighter prospect in the near future.

### THE FOOD PROBLEM

A major factor contributing to the unhappiness of man in our country to-day is the food problem. In our province the rice ration is reduced to well below physiological limit. It is true that wheat is occasionably added up in the meagre ration and that people are trying to get accustomed to varying diets. Dissatisfied people turn towards the black market, but the poor and the lower middle classes, not being able to face the black market, are unwillingly subjecting themselves to semistarvation. On the one hand there is inadequate procurement and ration, and on the other hand therefore hoarding of paddy and black marketing, occurring as a vicious circle, thriving on scarcity and corruption. This is a matter of no credit either to the Government or to the people. The result is malnutrition in the case of the vast majority and impoverishment or hand-to-mouth existence for others with moderate income.

### RURAL MEDICAL RELIEF

When I think of the villages, I am afraid that reconstruction of free India has started at the wrong end, for, these countless units that form the bulk and foundation of our land are still under neglect. It is true, as I said before, that we

cannot expect much in a short time. I am glad that slowly the Governments are directing greater attention as by the introduction of village Panchayat bills in the provinces. To bring the villages under better medical attention the Madras Government have introduced Primary Health Centres in certain villages, as a limited scheme for the present. These centres have paucity of medicines and common surgical instruments, and the medical men posted to the centres are able to do little justice to the patients attending except some thing of the nature of first aid or last aid due to lack of facilities. The village Vaidyas, Hakims etc., still flourish on ignorant masses as most of the villages are remote from hospitals. The large majority of qualified private practitioners seettle in towns for ovbious reasons. Why then should we complain that quacks flourish and endanger the lives that are threatened by disease? Rural medical relief has to be tackled by a number of ways and I venture to make the following suggestions. Firstly, the subsidised rural medical centres that were started by the late Raja of Panagal which are now in a dormant state must be revived. Times have changed and the cost of living index is over three times the pre-war level. Hence the same pre-war subsidies will not attract medical men, and as a matter of fact, even in the pre-war days the rural practitioner had no sufficient subsidy from the Local Board. He must be paid adequately and the rural dispensary must be sufficiently equipped. Secondly, the Primary Health Centres must be made selfcontained units and more of such centres must be started every year. Thirdly, the Taluk Head Quarters Hospitals must be improved in quality, personnel and bed strength. In this connection I shall give a simple dictum that if the Government can concentrate more of their attention on Taluk hospitals, the towns can take care of themselves. But will the Government act? I am afraid that in the face of the schemes to develop the City and District Head Quarters Hospitals, and in the present financial position, the Government will not follow this dictum. However, I feel it my duty to put the above suggestions before the Government. Let us hope and trust that the newly formed Central Council of Health will take early steps towards the problem.

The task of Rural Medical Relief is so immense that we cannot expect the Government to do everything to solve it, and we, men and women of the profession outside the Government can give a helping hand. Times without number, the young graduates, as they leave the portals of medical colleges are advised to settle in villages and lead a sacrificial, humanitarian life. It is easy to give advice, but the budding doctor, whose parents would have spent thousands of rupees over the professional course, would naturally desire to settle in a town to be sure of a decent earning. He cannot be blamed or dubbed as being too selfish. Now every town has a good number of qualified practitioners. They can organise medical relief in their own way in scores of villages lying over the inner and outer orbits of the town. What is required is co-operation and understanding and they can do it without detriment to their own interests. Each practitioner will get a turn once a week in visiting a group of villages and a record of patients and their progress is maintained which is handed over to the next doctor in turn. Thus a group of half-a-dozen villages can get medical attention twice or thrice in the week, depending on the number of practitioners in the town who work this scheme co-operating with each other. The poor will get free treatment while others will pay according to their means. When once an organisation is formed the details can be worked out. I wish that the district and town branches of our Association should start such model schemes and all the members and non-members can be persuaded to join it.

### Medical Education

Despite the vehement opposition of the medical profession throughout the country, the Health Ministers' conference held in August '50 accepted the recommendation of the Chopra Committee Report that a synthesised system of modern medicine, Ayurveda, Unani, Siddha, etc., be instituted as a curriculum and that diploma and degree courses should be started accordingly in the various States. Why have the authorities blinded themselves to well meant criticism and have gone off at a tangent? Is it that they have misconstrued that all those practising modern medicine have developed a bias, or hatred towards the indigenous systems, or is it that they agreed with the Chopra Committee that a synthesis is possible? If it is the former they can rest assured that we have all at one time or other sought aid of one or other of these systems and we have a reverence and due regard for these ancient systems. If it is the latter, all that I should say is that the Health Ministers have not studied the criticisms deeply before brushing them aside. They were warned that they must take the right step in this vital matter of medical education, as otherwise they would land themselves into a quagmire from which it would be very difficult to extricate themselves. The crux of criticism was that the ancient systems have therapeutic value while their diagnostic methods are obsolete and they have nothing to do with the basic sciences which are essential in the study of modern medicine. How then can they be synthesised for teaching raw under-graduates? For the last twenty years licentiates in Indian Medicine were produced out of a synthesised course in Madras. What was the result? The licentiate learnt neither of the systems satisfactorily, and in private practice he reverted mostly to remedies of modern medicine though without sufficient background, or knowledge of their use. If he has flourished it is a different matter. The art of medical practice is different from the science which embodies several branches, and even compounders with some experience of prescriptions do resort to practice, knowing the therapeutic actions of a few drugs and chemicals, though they know nothing of the science of disease. Hence, those that are likely to come out with degrees and diplomas in the synthesised course may flourish in practice by adapting themselves to some line, each in one's own way. Or as I expressed some time back and as others also think, they may turn into dangerous "Polypaths". Dr. T. N. Banerji in his presidential address at the All-India Medical Conference held at Sholapur stated "In the interests of the Indigenous systems of medicine we feel inclined to observe, that a course of four or five years is the irreducible minimum that is required for a student to be conversant with the Western system of medicine, or perhaps with the pure Indigenous systems as well. A large slice of this period will be taken away for training the students of Hygiene, Radiology etc. etc., but the result will be far from happy. I am afraid that the student can hardly be expected to be efficient in any system of medicine, after the termination of his 5 years course of studies. We, therefore, urge that instead of mixing the two systems of medicine from the very beginning and thereby making the confusion worse confounded in the

minds of immature students, let the synthesis come last. Those students who want to practise and be conversant with the Indigenous systems of medicine, should first graduate themselves from a college of modern medicine and then take up a course of training for a year, two years or more if necessary as a post-graduate student in one of the state research or post-graduate institutions in the Indigenous system of his choice."

I have to express my grave doubt whether the authorities, in starting the synthesised course for the under-graduates, have discriminated between the *art* and *science* of medicine. They may, with an anxiety of affording greater medical relief as an immediate step, use a short-cut of imparting training in hygiene, public health, sanitation and preventive medicine for any period to the Vaidyas, Hakims, etc. The public may be misled into the view that they are properly trained medical men. But the authorities have to remember that *basic medical education* can be imparted in one and only one way that is the scientific way and there can be no mixture and no confusion in the mind of the young trainee.

Every one is agreed on the recommendation of the Chopra Committee that a research can be instituted in the indigenous systems of medicine and that the wealth of therapeutic knowledge can be added to the armamentaria of the physician. The physiological, pharmacological and therapeutic effects of the drugs are to be thoroughly analysed by tests on experimental animals and on men. Their biochemical and bactericidal properties, and any injurious effects on vital tissues have to be determined and their dosages, possible routes of administration and indications, according to modern medical methods, have to be systematically listed. As our Health Minister Hon'ble Rajkumari Amrit Kaur remarked " ..... it is recognised that Ayurveda and Unani do contain valuable drugs and medicinal products and it is most desirable that such of these as can be tested by modern scientific methods and proved to be of value should be made available to all mankind." We can as well wait for a decade or more, if necessary while this is being achieved and our state Governments and Central Government would have subscribed to a pioneer work. The hasty introduction of a so-called synthesised course for the under-graduates cannot be approved by all right-thinking men of the profession.

Another aspect of this innovation is that it introduces different categories of medical men and women. The All-India Medical Association has been demanding that the basic medical qualification must be M. B. B. S. and this was also agreed to by the Indian Medical Council. The L. M. P. Course in Madras was rightly abolished, though in the confused atmosphere now prevailing there are mushroom suggestions, even in governmental circles, that it can be revived. The Madras Government have forestalled the resolution on synthesised course by starting the Graduate degree of College of Indian Medicine for which the Madras University rightly refused to give recognition. Is this not against the resolutions of the Indian Medical Council and the Indian Medical Association, and is the Government wise in re-introducing castes in medical profession ? We have already witnessed the recent strike of the G. C. I. M. students that they must be considered to be on a par with M.B., B.S. students in all their privileges and prospects. The students are right in their demand, pricked by an inferiority complex and the Government are wrong and are to be blamed for their creation. Even if the Government puts them on a par with the M.B., B.S. graduates, the distinctions of G.C.I.M., L.I.M. and M.B., B.S. cause a perpetual class struggle. Leaving the question of an unsynthesisable synthesis of medical *systems*, this is a retrograde step with regard to a uniform basic medical qualification and elimination of unhealthy strife in the medical profession. Let not prestige stand in the way of the Health Ministers in revising their decision and acting progressively and in consonance with well considered opinion of the Indian Medical Association and the medical profession and medical press in general in our country. The Madras Government will do well to convert the college of Indian Medicine into a pure research institution suitable also for training post-graduates in indigenous systems.

### The Bhore Committee Recommendations

Andhra Pradesh is about 63,000 Sq. miles in area and more than two crores in population. She has got approximately, 300 hospitals including dispensaries with a bed strength of about 2500, staffed by some 600 doctors. There are about 200 subsidised Rural dispensaries, besides a few Primary Health Centres. As per Bhore Committee recommendations, there should be 10,000 qualified doctors for Andhra, one for every 2000 of her population, their present number being only a little over 2000. She has one full medical college at Visakhapatnam and the other, the newly started one at Guntur, with preclinical courses only. It is obvious that Andhra Pradesh may require more than four medical colleges to produce one doctor for every 2000 of her growing population in about a period of fifty years. Rayalaseema should be provided soon with a medical college, while the Guntur medical college must be made a full institution. As stated by my predecessor Rao Bahadur Dr. B. Tirumalarao in his presidential address at Visakhapatnam in Feb. 1949, buildings with cheaper methods of construction on a ten or fifteen year basis must be resorted to by the Government in order to implement their schemes immediately, while permanent buildings may be constructed when the finances improve.

### Refresher Courses and Post-Graduate Training

There is no doubt that medical people should now and then take up refresher courses in order to be up-to-date in their knowledge and treatment and it should not be considered as a waste of time, as the benefit derived thereby is immense in practice. The Madras Government was sympathetic enough to start these courses at Madras and Vizag and I hope that they will continue them every year. There seems to be some dissatisfaction for those who attended them that not much was taught and learnt, but they have to remember that the clinical professors have got to adapt themselves to the requirements of practitioners and this new and beneficial line may take some time in bearing fruits to the satisfaction of all. The public and the Government who expect so much of selfless service from medical men would collect no fees for Refresher courses both from private practitioners and service doctors.

We feel glad that the Government of India have set up an up-grading commission and that the Anatomy and Venereal departments in Madras Medical College are to be up-graded. I hope that the Madras Government will set apart sufficient funds for the purpose and with the help of the Government

of India, will establish the up-graded departments immediately. I wish that they improve other departments in Madras and Vizag with the same view. I trust that young aspiring graduates will take the benefit of the up-graded departments. We are also glad that the Government of India and the State Governments have instituted over-seas scholarships and deputations. I am sorry, however, that the Madras Government frequently interferes with the selection of candidates by the committee of the principals and those who go for interview at New Delhi before the Final Board of selection are not the same persons as recommended by the committee of principals. I hope the Madras Government will rectify this and give no room for complaints of favouritism.

### Madras Medical Service

While the Madras government have abolished the rank of Sub-Assistant Surgeons as per demand of the licentiates, they have only substituted it by class II Assistant Surgeons. The distinction of Class I and II among assistant surgeons must be removed and there must be a uniform basic pay for all. Why should a medical graduate draw less pay and suffer an inferior rank as Class II Assistant Surgeon or as a Demonstrator in a college ? Did he not undergo the same trouble in his studies and spend as much as one who is selected to class I ? I have to suggest that the earlier this injustice is removed the better. The method of selection also to the Madras Medical Service is far from satisfactory. It is done by the Madras Public Services Commission, with one doctor who may be a professor or principal included in the panel and marks are given to the candidates at interview. The communal G. O. is also virtually followed. On what basis the marks are given is not known. The selection must be made by a expert medical committee. I cannot help remarking that Government jobs are considered by communal minded men as loaves and fishes to be equally distributed, regardless of talent, hard work, honesty and a sense of responsibility and devotion. Selection also must be based on the inclination of the candidates to particular branches of work and the right spirit and earnestness to serve suffering humanity.

### Our Association (I. M. A.)

It is a painful fact that the majority of medical men and women in Andhra Desa have not joined I.M.A. and consequently the membership strength of the Andhra Provincial Branch under whose auspices this conference is now being held is about 600, though there are more than 2000 qualified medical people in the Andhra area. I appeal to every one of them to treat the I. M. A. as the parent organisation and join it forthwith. We must strive our level best to develop I. M. A. as strong as B. M. A. and make it powerful and the only mouthpiece of medical profession in England. Then only the Government will, I hope and trust pay due heed and not have the courage to reject the recommendations of the I. M. A. The prevailing outlook with some doctors, I am afraid may be that there is no positive benefit for them, if they should join I. M. A. This is wrong ; ultimate benefits are surely there, and it is my urge and appeal that they should enlist themselves as members just to strengthen the organisation and the hands of their brethern who are working hard for the cause of the noble profession in a selfless way.

It is my strong urge that our Government should withdraw the order prohibiting their medical officers from holding the office of Secretary or Treasurer of the Medical Association which is only an academic body for all intents and purposes.

### Amalgamation of A.I.M.L.A. & I.M.A.

It has been a vexed question dragging on for years. It is regrettable that all licentiates have not recognised yet the serious folly that they are subscribing to the division of the profession in continuing the A.I.M.L.A. separately. It was the British rulers who encouraged its existence. Our national Government have definitely expressed more than once their discountenance against its continuation any longer. Being myself a licentiate, it is my honest feeling that the A.I.M.L.A. has outlived its usefulness long since, neither has it the encouraging support of our Government, nor the profession at large. The majority of licentiates do not like its separate existence at all. Its funds may profitably be earmarked as Welfare Fund and utilised for the education of the deserving children of the licentiates, and the I.M.A. have already given their undertaking to that effect, as I have been aware. The move of some of the licentiates to change the name of the A.I.M.L.A. into Bharat Medical Association with a view to enabling the graduates to join it does not contribute to the progressive side of the medical politics in India. The sooner it is wound up the better, as it is exhibiting unhealthy complexes only and nothing else. It is known to all that, it is the I.M.A. that our popular Government have rightly recognised for all purposes. I am fully confident that my brother licentiate leaders for whom I hold great respect, will lose no time to look at the things in their proper perspective and bring about the much desired amalgamation of A.I.M.L.A. with I.M.A. and thus help to promote unity in the profession and achieve the ideal of establishing one consolidated medical organisation for India. Now it is time that the Medical Council of India would take up the question and include the licentiates in its register in an alphabetical order on par with the graduates, and I hope our Union Government will press for the same.

### "Public Health"

Apart from Government's duty that steps should be taken for organising adequate and efficient Public Health Service, we, the medical men and women of the province must play a great part in improving the general health of Andhra Desa. The duty of the medical practitioner is not only the cure and prevention of diseases but also raising the general standard of health of the individual. He has the first hand knowledge of the bad conditions of external environment, of common practices inimical and opposed to personal hygiene of what is for the time being the prevalent disease, and it might well be considered as a part of his duty, though not always compulsorily imposed, to place all such knowledge at the disposal of the Public Health Officer of that area or locality. I recommend that a harmonious organisation of their combined work and effort within a single sphere should be sought and established, and this should be one of the outstanding features of any practical National Health Service. Scientific investigations have shown the importance of nutrition as a factor in health. Some diseases prove to be wholly and others partly due to deficiencies of protective food substances. Measures against malnutrition are, therefore, of vital importance and we can on our part educate our patients as to what food to take and how to live better within their means.

### Honorary Service

I recommend strongly that each speciality, as Eye, E. N. T., Venereal etc., at the district hospitals should have compulsorily two hony. medical officers, styling one of them as senior officer. The administration should not interfere with their routine work. Hony. Medical Officers also should be privileged by rules to submit confidential reports of the amenities offered to them by the administration and the difficulties met in discharging their duties to the Surgeon-General every year independently of the D. M. O. or the Superintendent of the hospital.- I am sure this will go a long way to help creation of real harmony and turning out useful work. On the one hand our Health Minister wants to economise the medical department by increasing the net work of honorary system, on the other hand he wants the hony. men to uphold the ideals of service. The hony. system has already become so expanded that young aspirants of the medical profession are attracted to it, and naturally there will be grievances from these with regard to the facilities given for their work, the differences that are bound to arise between the paid and the honoraries, the adverse criticisms of the paid superiors and the like. The Government should recognise the same and feel obliged to rectify them. If the Government sincerely want to improve the system, as they want to expand it, they must agree to pay a reasonable honorarium to the Hony. Asst. Medical Officers in particular. With regard to physicians and surgeons of professional reputation it is left to them to accept or reject the offer of hony. jobs when the Government approach them on 'bended knees' as stated by the Health Minister at the Hony. Medical Officers' Conference held at Madras last year.

### Drug Industry

The Government should extend their financial support to the drug industry sponsored by Indians with Indian capital. It is happy that the Drugs Control Act is in force and the flooding of the Indian market with spurious drugs has become an impossibility. It is time that the members of the profession should well interest themselves in the manufacture of our essential requirements. Under the present conditions, the Government should freely import such of the drugs as Penicillin and Streptomycin etc. and make them available to the profession at reasonable prices It is our duty to give preference to Indian manufacturers over foreign products. Doctors should also try, experiment upon and use the new medicines manufactured by the Indian concerns, and if they find them lacking in some thing they should not discard them as useless, but voluntarily suggest ways and means to the manufacturers for their improvement. Thus, they may exercise a check over the productions and bring their level to a much higher standard of efficiency, with the ultimate good result that the profession need not depend on the import of foreign drugs any longer. To overcome and root out black-market and exploitation, I suggest that the members of the profession should start drug stores in every town under the Co-operative Department as at Guntur where a Co-operative Medical Store managed by the doctors of the locality has been working successfully since 1939.

### Conclusion

Men of all professions must devote part of their energies towards national reconstruction and assist the Government, so that our country will advance towards the goal of perfection as an independent nation. This may be a question of not a decade or two but more. The citizens are not to think of their personal interests alone but must be prepared to suffer the present hardships and the toil involved in performing this great and noble task. They cannot and should not look for immediate rewards, but the coming generations will derive the benefit. This philosophy of life should be recognised by all and put into practice for the salvation of the nation, and we, the members of the medical profession shall play our part quite willingly and well.

## RESOLUTIONS PASSED

The following resolutions were passed at the Rajahmundry Conference, 1951 :—

1. This Conference records its sorrow at the demise of Dr. P. Rajarao of Rajahmundry, Dr. K. S. Ray of Calcutta, Major M. G. Naidu of Hyderabad and Dr. C. R. Reddi.

2. This Conference recommends to the University to amend their medical inspection rules suitably to permit the licentiates to be college medical officers and inspectors.

3. This Conference resolves to urge on the Government of Madras to start as early as possible a Medical College in any one of the districts of the Rayalaseema.

4. This Conference urges on the Government to abolish the category of Class II Assistant Surgeons altogether.

5. This Conference while thanking the Government for a token provision in the budget, urges on the Government to take steps to start the 3rd M.B. Class in the Guntur Medical College by the beginning of the next academic year i.e. July, 1951.

6. This Conference urges on the Government to subsidise adequately primary medical centres in rural areas voluntarily run by the co-operation of at least two medical practitioners.

7. This Conference strongly deprecates the manner in which the Central Bank of India Ltd. has demanded a comission of 40% from its medical advisers, from the remuneration they are entitled to, as per the award. Acceptance of such conditions by a practitioner is against professional ethics.

8. This Conference urges on the Government to suitably amend the rules for election made under the Madras Medical Act 1914 to provide for a judicial tribunal to deal with election complaints if they deem it necessary ;

b) and to immediately cancel the rules made some time ago authorising the Government to take away the powers of the President or the Registrar, at their discretion and delegate them to some other officer like the Surgeon General.

c) this Conference strongly protests against the reported move to amend the Act, to provide for an ex-officio—instead of an elected president for the Madras Medical Council.

9. This Conference urges on the Andhra University—

a) to take steps to organise post-graduate training and research taking advantage of the facilities available at the Andhra Medical College and the K. G. Hospital.

b) this Conference suggests to the University to take some suitable, experienced and competent medical practitioners on the Boards of Studies and Examiners.

**AJMER BRANCH**—The 15th annual general meeting of the branch was held on the 1st April 1951 at the Civil Hospital with Dr. C. H. Primlani in the chair. After the report and accounts were passed, Dr. Primlani and Dr. S. P. Sen were elected as president and secretary of the branch for the year 1951-52.

**ALLAHABAD BRANCH**—At the last annual general meeting of the branch held on the 3rd April, 1951, Dr. Brijraj Kishore and Dr. S. K. Mukherji were elected as the president and the secretary of the branch for the year 1950-51.

**AMRAVATI BRANCH**—At the last annual general meeting of the branch, it was disclosed that the branch had 48 members which constituted about 95 per cent of the qualified doctors of the town. Scientific papers were read and clinical discussions were held regularly at the monthly meetings.

**ASANSOL BRANCH**—At a meeting of the branch held on the 30th March 1951 with Dr. B. N. Roy in the chair, the members noted with great concern, the recent order of the Government of West Bengal for reduction of beds in the local government hospital from 52 to 32. It was regretted that before the proposed central regional hospital was established by the Government of India from the Coal Miners' Welfare Fund, such a step would add to the sufferings of the workers and the local people.

The association appealed to the Government of West Bengal for increasing the number of hospital beds and for the provision of a separate tuberculosis clinic with x'ray facilities, at Asansol.

The branch also opined that the system of issue of permits for streptomycin etc. by the Civil Surgeon alone is not working to the benefit of the public. Some responsible medical men should also be entrusted with the work.

The branch also requested the authorities of the Asansol Municipality not to reduce their meagre grant to the Leprosy Clinic at Domohani Road, Asansol.

**ASSAM PROVINCIAL BRANCH**—A meeting of the Working Committee of the branch was held on the 16th June, 1951 with Dr. H. K. Das in the chair. 9 members were present.

The committee decided to invite the Working Committee of the I.M.A. to hold its October meeting on the 14th and 15th October 1951 at Gauhati. It was also decided to hold the 3rd Assam Provincial Medical Conference at Gauhati on the 15th, 16th and 17th October, 1951.

The Secretary informed the committee that new local branches were duly formed at Golaghat, Karimgunj, Sibsagar and Tinsukia and attempts were being made to form another new branch at Labac.

**BENGAL PROVINCIAL BRANCH**—The second annual meeting of the Bengal Provincial Council, under the new rules, was held on the 17th February 1951, with Dr. A. D. Mukharji in the chair. 45 members were present.

At the outset, the members condoled the sad deaths of Major M. G. Naidu of Hyderabad, a past president of the I.M.A. and Dr. P. S. Bhattacharya, a past president of Tarakeswar branch.

After the annual report and the audited accounts were adopted and the budget passed, Dr. A. C. Ukil and Dr. P. K. Chatterjee were declared as duly elected president and secretary of the provincial branch for the year 1950-51.

Before the meeting terminated Dr. A. D. Mukharji who was the president of the branch for the last two successive years, thanked all concerned for their help and co-operation.

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At another meeting of the branch council held on the 5th March, 1951 with Dr. A. C. Ukil in the chair, 35 members were present.

The members condoled the sad death of Dr. S. Majumder of Naihati.

The Secretary informed the house that a new branch was formed at Balurghat with 20 members. While the standing committee and sub-committees were being formed, the secretary informed the house that Dr. P. K. Guha expressed his inability to accept membership or convenership of any committee or sub-committee, on personal grounds. The council then elected 21 members to the provincial working committee and constituted several other standing committees and sub-committees.

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A meeting of the working committee of the Bengal Provincial Branch was held on the 17th April, 1951 with Dr. A. C. Ukil in the chair. 24 members were present.

The members condoled the sad death of Dr. Nagendra Nath Bhattacharya (Sr.) a past president of the Branch.

The secretary informed the committee that the Asansol branch had been revived with 19 members. As publication of the first edition of the Bengal Medical Directory was being delayed unduly, it was decided to communicate with Dr. P. K. Guha, the Editor of the Directory.

The committee approved the nomination of Dr. K. K. Sen Gupta as a representative of this branch to the governing body of the School of Tropical Medicine.

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At an adjourned meeting of the working committee of the branch held on the 24th April, 1951 with Dr. Ukil in the chair, 20 members were present.

The secretary informed the house that the medical education and health services committee had formulated a report on the question of pay, status etc. of medical officers. The committee decided to circulate the report to local branches.

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At an emergent meeting of the working committee of the branch held on the 3rd May, 1951 with Dr. Ukil in the chair, 21 members were present.

The committee considered the question of nomination of a member to the visiting committee of the Nilratan Sarkar Hospital and decided to refer the matter to the Calcutta branch for suggestions.

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At another meeting of the working committee of the branch held on the 15th May, 1951 with Dr. Ukil in the chair, 22 members were present.

The members condoled the sad death of Dr. K. Bhaduri, C.M.O. of the Port Commissioners Hospital.

The secretary informed the members that a new local branch was formed at Bongaon with 5 members and the Malda branch was revived with 16 members.

**BONGAON BRANCH**—At a meeting of the branch held on the 20th May, 1951 with Dr. J. R. Dhar in the chair, Dr. Dhar was congratulated on his election as secretary of the Bongaon College (Dinabandhu Mahavidyalaya).

**CALCUTTA BRANCH**—The 4th meeting of the branch committee for the year 1950-51 was held on the 10th April, 1951 with Dr. J. C. Banerjea in the chair. 27 members were present.

The Committee strongly disapproved the action of the Government of West Bengal in opening the Venereal Diseases Out-patient Department of the Calcutta Medical College Hospital in the basement rooms of the Eden Hospital in spite of the unanimous recommendations and protests of the visiting committee of the medical college group of hospitals. The Committee agreed with the views expressed by Dr. A. K. Sen as a representative of the association to the said visiting committee and was of opinion that the recommendations of the visiting committee should be accepted by the State Government.

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At the 5th meeting of the branch committee held on the 8th May, 1951, with Dr. Banerjea in the chair, 21 members were present.

Dr. M. Biswas informed the committee that Rs. 150/- has been received as library grant from the Corporation of Calcutta

and the library committee proposed to spend about Rs. 2500/- for the purchase of books, during the current year.

Requested by Dr. M. K. Roy Choudhury, Dr. A. K. Sen representative of this association to the local telephone advisory committee explained the mode of working of the advisory committee.

Dr. K. C. Chakraborty was nominated by the branch as its representative to the Nilratan Sarkar Medical College Hospital Visiting Committee.

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At a special meeting of the branch committee held on the 17th May, 1951 with Dr. Banerjea in the chair, 19 members were present. The question of pay, prospects and the status of medical personnel in West Bengal was considered in detail.

The Committee recommended that the remuneration of the part-time visiting physicians, surgeons and specialists should be Rs. 350/- to Rs. 500/- p.m. according to qualifications and experience. The committee also recommended that the starting salary of the whole-time and non-practising demonstrators and registrars should be Rs. 350/- p.m.

**COIMBATORE BRANCH**—A meeting of the branch was held at Siruvani on the 22nd April, 1951 with Dr. M. G. Nair in the chair. 54 members attended the meeting.

Dr. Y. P. Vasudevan spoke on the preventive aspects of tuberculosis. He also outlined the plan for starting a tuberculosis hospital at Coimbatore, for which Dr. C. Nanjappa, promised the financial assistance of the local municipality.

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At another meeting of the branch held on the 26th May, 1951 with Dr. Nair in the chair, about 72 members were present.

Dr. C. Gopalan spoke on the "clinical uses of newer B Vitamins". Dr. M. Krishnaswamy demonstrated an improvised machine of "sphygmograph".

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The next meeting of the branch was held on the 30th June, 1951 at the local military hospital with Dr. Nair in the chair. About 90 members were present.

A sub-committee, with powers to co-opt, was appointed at this meeting to devise ways and means for the construction of a tuberculosis hospital at Coimbatore.

Dr. V. G. Govindan Nair spoke on "common skin diseases". This was followed by a film shown on "Aureomycin" by Messrs. Lederle & Co.

**DOOARS BRANCH**—The last annual general meeting of the branch was held on the 18th March, 1951. Drs. G. N. De and S. N. Das were elected as the president and the secretary of the branch for the year 1950-51.

The secretary reported that the branch had 79 members at the beginning of the year 1949-50 and 144 members at the end of the said year.

The members resolved that the Dooars branch being the only representative body of the members of the medical profession of the area, the D.T.G.I.E.A. and T.G.I.E.A. should consult this branch while dealing with medical and health matters.

**GHUGUDANGA BRANCH**—At a meeting of the branch held on the 27th May, 1951, with Dr. S. Sarkar in the chair, 23 members were present.

Dr. D. Poddar spoke on "Puerperium, its management and complications."

**GOLAGHAT BRANCH**—An inaugural meeting of the members of the medical profession of Golaghat in Assam was held on the 1st May 1951, with Dr. P. R. Das in the chair.

It was resolved to form a local branch of the I.M.A. at Golaghat and Dr. Das was elected as the president of the branch. Drs. C. D. Choudhury and A. Rashid were also elected as the joint honorary secretaries.

**GORAKHPUR BRANCH**—At a special meeting of the branch held on the 25th April, 1951 with Dr. P. D. Srivastava in the chair, 6 members were present.

The members condoled the sad and sudedn death of Dr. J. N. Srivastava, a senior member of the branch.

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Another meeting of the branch was held on the 2nd June, 1951 with Dr. Srivastava in the chair.

Dr. N. R. Banerjea spoke on his observations on the treatment of malaria cases with paludrine. Discussions were also held regarding a case with enlarged spleen and lung affections.

**JAGATDAL BRANCH**—A meeting of the branch was held on the 15th May, 1951 at the Bhatpara municipal hall with Dr. S. Chakravorty in the chair.

Dr. A. Chakravarty spoke on "intestinal amœbiasis".

**KHAMGAON BRANCH**—A meeting of the branch was held on the 8th April, 1951 with Dr. A. V. Khare in the chair.

The members passed a resolution expressing their concern on the retrograde and unhealthy step taken by the Madhya Pradesh State Government by instituting the "three-year medical course", in the year 1948.

**KANPUR BRANCH**—A special meeting of the branch was held on the 28th June 1951.

The members condoled the sad death of Major N. D. Banerji.

**MADURA BRANCH**—A meeting of the branch was held on the 16th April, 1951 with Dr. M. Rajan in the chair.

The members discussed the insurance scheme submitted by an insurance company.

Dr. J. C. Savarirayan spoke on "surgery in the United States".

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At another general meeting of the branch held on the 23rd June, 1951 with Dr. Rajan in the chair, 45 members were present.

The members discussed the details of the building plan. The members passed a resolution requesting the Government to extend to the medical officer attached to the local authorities and municipal dispensaries facilities to attend the meetings of the I.M.A. They also recommended that the period of attendance at these meeting should be considered as 'on duty'.

Dr. M. G. Varadarajan spoke on 'X'Ray therapy in the treatment of non-malignant diseases'.

**MALABAR BRANCH**—A meeting of the branch was held on the 28th April, 1951 with Dr. N. Achuthan in the chair.

Dr. C. K. Menon spoke on the 'scope of radiology in general practice'. Dr. A. G. Menon demonstrated a few interesting cases.

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At another meeting of the branch held on the 9th June, 1951 with Dr. Achuthan in the chair, Dr. V. G. Nair spoke on 'modern conception of syphilis and its treatment'.

**PORBANDAR BRANCH**—A meeting of the branch was held on the 27th May, 1951 with Dr. H. H. Chavda in the chair, 19 members were present.

Drs. K. W. Waghela and K. B. Shah spoke on 'artificial pneumothorax' and 'coma' respectively. Dr. B. D. Jhala spoke on a case of 'splenectomy'.

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At another meeting of the branch held on the 27th June 1951 with Dr. Chavda in the chair, 22 members were present.

As Dr. K. W. Waghela, had to leave the place for 2 years, his services as secretary of the branch were praised and Dr. B. D. Jhela was elected as secretary of the branch.

**PUDUKOTTAH BRANCH**—A meeting of the branch was held on the 5th May, 1951 with Dr. V. S. Iyyer in the chair.

The members passed a resolution of congratulation on the appointment of Dr. K. Vasudeva Rao, M.D., F.R.C.P., T.D.D.(W), as the Director of Medical Services, Madras State.

Major G. A. Naidu then spoke on 'skin diseases in general practice'.

**RAMNAD BRANCH**—A meeting of the branch was held on the 10th May, 1951.

The members regretted that the President and the Vice-president had to tender their resignations as they were not permitted by the Government to continue as office-bearers of the I.M.A.

Dr. M. G. Jones was elected as the new president and she thanked the members on her election.

**SHAHABAD BRANCH**—At a meeting of the branch held on the 15th April, 1951 with Dr. V. Shankar in the chair, 20 members were present.

The members condoled the sad death of Dr. A. N. Sarkar.

The members decided to try to have a building for the branch. The members also resolved to organise a body show in the last week of May, 1951.

The Committee requested the Government to allot one seat of the nominated Arrah Municipal Board to this branch of the I.M.A.

**SOUTH INDIAN PROVINCIAL BRANCH**—A meeting of the branch council was held on the 8th April, 1951 with Dr. U. Krishna Rao in the chair. 43 members were present.

At the outset, the council condoled the sad death of Major M. G. Naidu, a former president of the I.M.A.

The council opined that no sales tax should be levied on the fees collected by the radiologist for taking x'ray photographs.

The council provisionally accepted the invitation of the Malabar branch for holding the next provincial conference at Calicut on the 8th, 9th and 10th September, 1951. The council resolved to send a message of good will on the formation of the "Mysore Provincial branch".

**TANJORE BRANCH**—The 15th annual meeting of the branch was held on the 25th March, 1951 with Dr. K. V. Rao, in the chair. About 150 members were present.

The members were of opinion that it would be desirable to have a medical college in the Tanjore district. The members also recommended that an well-equipped laboratory should be established in every district.

In the presidential address, the president informed the members that the Government of Madras were trying to start two medical colleges, one in Andhra-desa and another at Madura.

Dr. R. Subramanian spoke on 'Congenital heart diseases'.

In the afternoon session of the meeting, at the outset, the members condoled the sad death of Dr. M. S. Raghavendra Rao of Manalmedu.

Dr. N. Vaidyanathan and Dr. N. R. Subramaniam were then elected as the president and the secretary, respectively, for the year 1951-52.

Dr. D. S. Iyer spoke on 'abdominal pain, its diagnosis and treatment'.

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At another meeting of the branch held on the 29th April, 1951, 45 members were present.

Dr. Vaidyanathan the president having been transferred, Dr. E. K. Menon was elected as the president.

Dr. M. S. Narayanam spoke on 'anæmias' hæmatology and therapeutics'.

The branch resolved that the Government of Madras should reconsider their order no. M.S. 3385/Health dated 8-4-46 and should permit medical officers in local board services to avail themselves of Sundays and all public holidays, with necessary permission.

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The June meeting of the branch was held on the 24th June, 1951, with Dr. Menon in the chair. 30 members were present.

Dr. K. I. Thommi spoke on some emergency conditions.

**TINNEVELLY BRANCH**—A meeting of the Tirunelveli branch was held on the 25th February, 1951 at Papanasam with Dr. K. R. Ayyar in the chair. 32 members were present.

The members had a stroll in the picturesque surroundings of the Hope lake before the meeting.

Dr. R. Subramaniam spoke on 'artificial pneumothorax'.

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Another meeting of the branch was held at Palayamkettai, on the 31st March, 1951 at which 42 members were present.

Dr. T. Alwar spoke on 'idiopathic hypopiesia'.

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The next meeting of the branch was held on the 28th April, 1951 at Tuticorin and 31 members were present.

The members condoled the sad deaths of Dr. D. Nallathambi Pillai and Dr. Mrs. E. Bernard Carr.

The resignation of Dr. K. M. Menon, the president, was accepted, with regret.

Dr. V. Kuruvilla then spoke on 'acute abdomen'.

The secretary thanked the Tuticorin Medical Association for excellent arrangements made for the meeting.

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At another meeting of the branch held on the 26th May, 1951, at Palayamkettai, 37 members were present. Dr. K. Rama Ayyar was elected to the chair.

Dr. Ayyar spoke on 'diseases of the testes'.

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The next meeting of the branch was held on the 30th June, 1951 at Palayamcottai. 40 members were present.

The members promised to pay an additional sum of rupees ten each for holding a joint conference of the medical associations of the southern districts.

Dr. G. Subramaniam then spoke on 'recent advances in therapeutics'.

**UTTAR PRADESH STATE BRANCH**—The third meeting of the working committee of the branch was held on the 29th March, 1951 with Dr. A. C. Banerjea in the chair. 10 members were present.

At the outset the members condoled the sad deaths of Dr. M. G. Naidu, a past president of the I.M.A., Capt. A. P. Misir (of Banaras) an ex-president of this state branch, Dr. H. L. Khorana of Dehradun and Dr. Baij Nath Varma of Lucknow.

The members resolved to put forward the difficulties of doctors in going to villages in larger numbers. The budget speech of the honourable health minister of the state was discussed in this connection.

The members appealed to the local branches of the State to contribute to the building fund of the association.

The members discussed the question of services of medical practitioners at night in connection with a letter from the Director of Medical and Health Services of the State on the subject.

The members welcomed the response of the professors of the medical colleges in the State regarding post-graduate lectures in the mofussil and requested the secretary to arrange a programme for the same in consultation with the local branches and the professors concerned.

**VIJAYAWADA BRANCH**—A meeting of the branch was held on the 25th October, 1950 at which 23 members were present.

The members condoled the sad death of Dr. K. S. Ray, the Editor of the *J.I.M.A.*

Dr. P. S. Giri then spoke on 'clinical methods in the diagnosis of liver diseases'.

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At another meeting of the branch held on the 24th December, 1950, 25 members were present.

The members condoled the sad death of Honorable Sardar Patel.

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At the meeting of the branch held on the 28th January, 1951, 28 members were present. Dr. Giri spoke on 'eosinophilic lung'.

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The monthly meeting of the branch for the month of March, 1951 was held on 31st March, 1951 at which 24 members were present.

Dr. P. R. Sastry spoke on 'influenza'.

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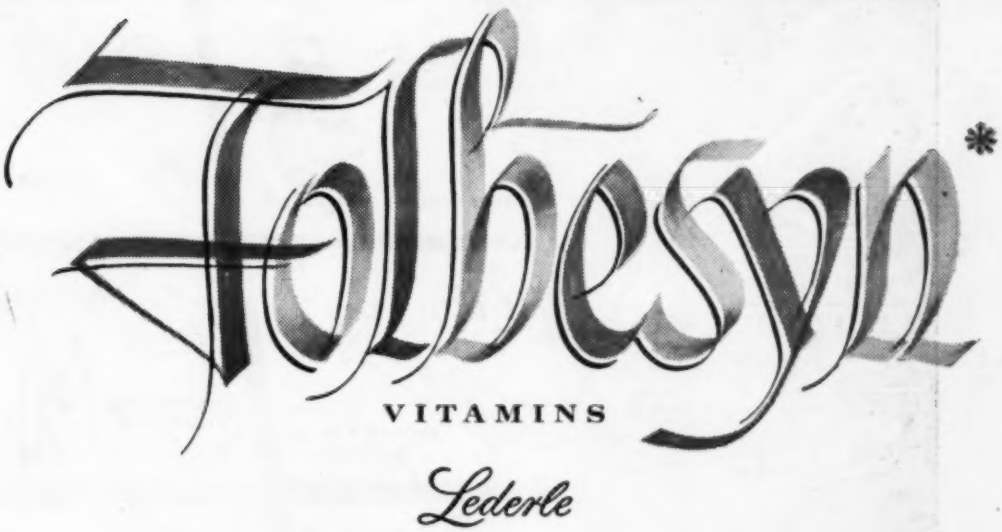


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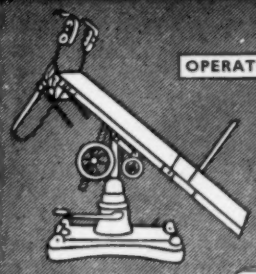
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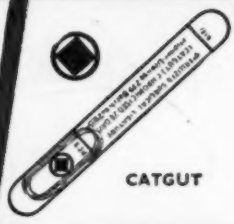
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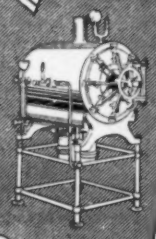
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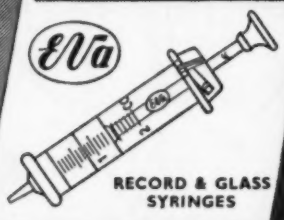

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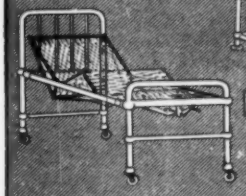
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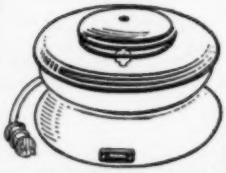
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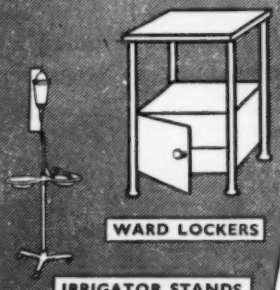
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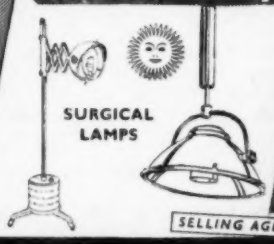
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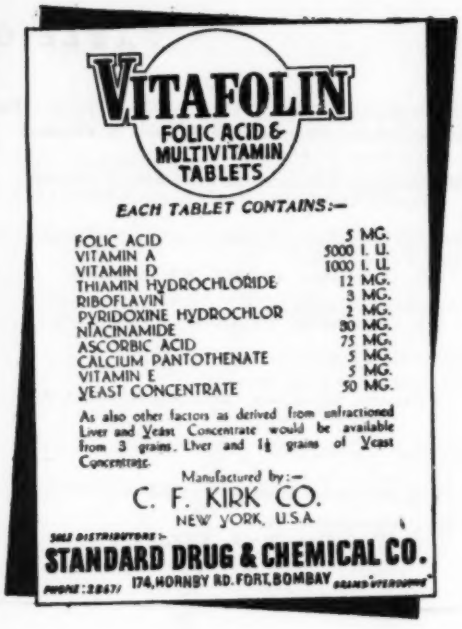
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| Extract Red Bone Marrow, Glycerinated | 2 c.c. |
| Ferric Ammonium Citrate .. | 0·87 Gm. |
| Malt Extract, non-diastatic .. | q.s. |

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**(Sodium Paraamino Salicylate)**

The introduction of AMINOPAR manufactured by the world renowned Union Chimique Belge ensures the supply of a product which is guaranteed to be of the finest possible quality at the minimum rate.

**GRANULES**
*66% P.A.S. Chocolate brown. In tins of 100 gms.*

**TABLETS**
*0.5 gm. Chocolate brown. Bots. of 1000 and 100 tablets.*

# TIOCARONE

**(Thiosemicarbazone)**

*Limited supplies in the following packing are available:*

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is a recently developed synthetic product known chemically as Para-acetylaminobenzaldehyde Thiosemicarbazone. It shows a powerful action against Koch's bacillus both in vivo and in vitro, and clinical results obtained with it are most promising. TIOCARONE can be used in conjunction with Dihydrostreptomycin or P.A.S., and it is reported that this association improves the activity of each drug separately.

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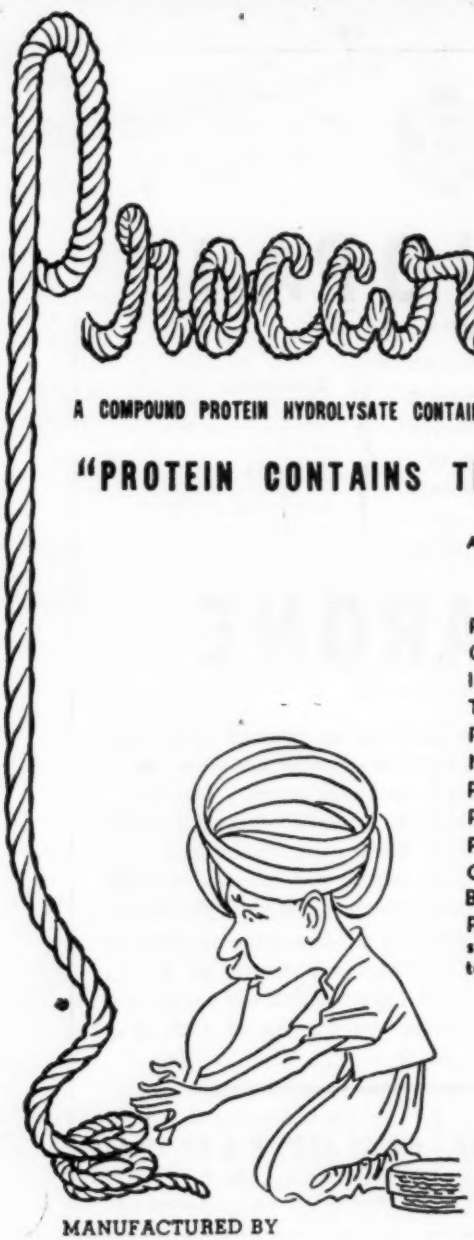
A COMPOUND PROTEIN HYDROLYSATE CONTAINING B COMPLEX FACTORS & MINERALS

"PROTEIN CONTAINS THE MAGIC OF LIFE"

AVAILABLE IN SEALED BOTTLES OF 4 OZS.



100 grams will supply :

| | |
|---|---|
| Protein (90% Soluble) | 49 grams |
| Carbohydrates | 40 grams |
| Iron (300% MDR) | 30 mg. |
| Thiamine (1500% MDR) | 15 mg. |
| Riboflavin | 7.5 mg. |
| Niacin | 40.0 mg. |
| Pyridoxine | 2.5 mg. |
| Folic Acid | 2.0 mg. |
| Pantothenic Acid | 12.5 mg. |
| Choline | 125.0 mg. |
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Plus other B Complex factors and minerals such as Calcium & Phosphorus etc. natural to yeast, liver and milk.

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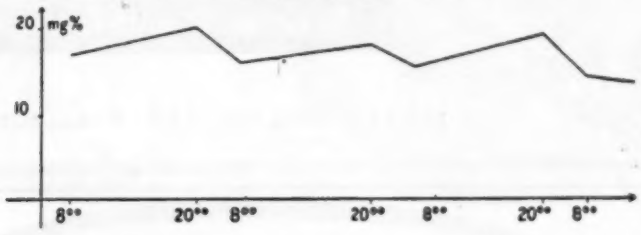
**(Boehringer, Mannheim, W. Germany)**

**INTRODUCED IN INDIA, IS**

# "P. A. C."

"P.A.C." combines in an ideal form, PAS and Calcium. Besides the fact that it is exceedingly well tolerated, and brings about an early improvement of clinical symptoms, such as nightsweats, temperature and expectoration, with a tendency of increase in weight, it offers a number of other advantages in the treatment of T.B., one of the most outstanding being the absence of diarrhœa which is frequently found after administration of other salts of PAS.—

Administration of Neutral Calcium Para-Amino-Salicylate ("P.A.C.")
1 G of "P.A.C." 4 times a day—Total in 3 days=12 G.




The 'graph' reproduced above shows that 4 G = 8 tablets of "P.A.C." per day will result in, and maintain, a remarkably high blood level with extensive bacteriostatic reaction.

The daily dose of 4 G of "P.A.C." taken by the patient, was administered in 4 equal doses spread over the **day.** No "P.A.C." was given at night. Yet the following morning, the blood concentration was found to be in the neighbourhood of 15 mg%!! The significance of this for both doctor and patient need hardly be emphasized.

"P.A.C. is 100% active substance, each 100 G corresponding to 88 G of PAS and 12 G of Calcium.—It keeps stable in all climatic condition.

**THE COST OF TREATMENT OF A T.B. CASE CAN BE REDUCED TO THE MINIMUM BY THE USE OF "P.A.C."**

*Doctor, while prescribing neutral calcium para-amino-salicylate write*
***"P.A.C."** . . . . so easy to remember.*

*for further particulars write to :*

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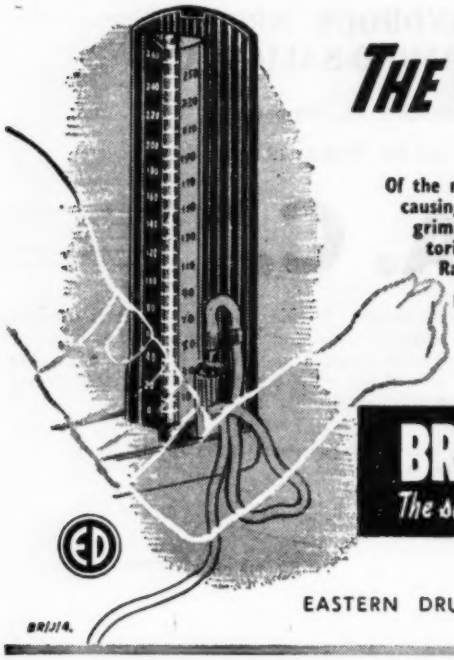
PHONE: 42859.



# THE GRIM PICTURE

Of the many types of heart diseases capable of causing death hypertension certainly presents the grimmest picture. It can be treated satisfactorily with Bromo-Raulfin, the standardised Rauwolfia compound.

Bromo-Raulfin reduces the elevated blood pressure and maintains it at the lowered level, gives the patients a feeling of well being and is free from all toxic effects.

**BROMO-RAULFIN**

*The successful hypotensive agent*

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*The liver-yeast-iron tonic*

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# penicillin nonad tulle

In all branches of surgery, Penicillin Nonad Tulle has been welcomed as an effective bacteriostatic dressing for operation wounds, including those of eyes, ears and nose, and those of skin grafting.

This non-adherent, sterilized gauze dressing of wide mesh is impregnated with an emulsifying base containing 1,000 units of penicillin per gramme.

Dressings of Penicillin Nonad Tulle are easily and painlessly removed without destroying the granulation and epithelial tissues in process of formation.

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*In tins, containing 40 pieces each 2"× 2", 10 pieces each 4"× 4", 40 pieces each 4"× 4", or a strip dressing 4"× 2 yds.*

ALLEN & HANBURYS LTD
( INCORPORATED IN ENGLAND )
CALCUTTA BOMBAY

# MAG-MAG

A quick, gentle, hygroscopic method of treatment to draw out toxins from affected parts. Accelerates healing.

**INDICATIONS:-CARBUNCLES, ABCESSES, BOILS, GLANDULAR SWELLINGS AND ARTHRITIS.**

Packed in screw cap pots sufficient for several applications.

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# SU-MAG

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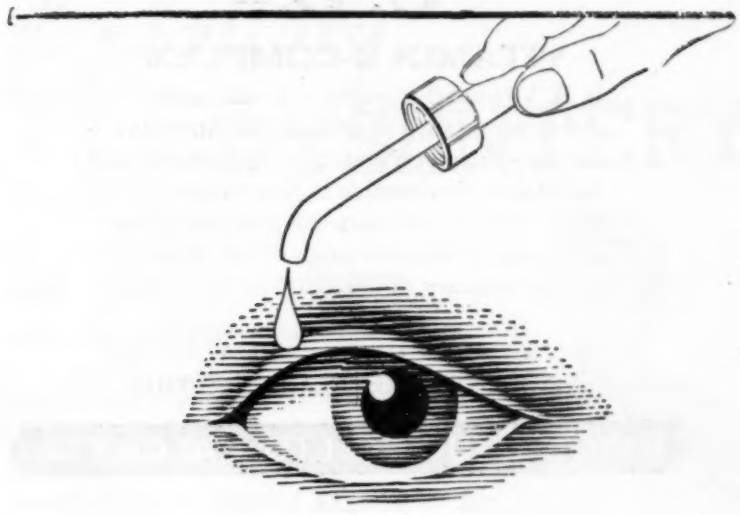

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A stable colloidal silver suspension of established importance in the treatment of inflammatory conditions of the eye, ear, nose and throat. The unique non-irritant and non-staining properties of the solution ensure perfect safety to all epithelial tissue even when used frequently and for long periods. In the eye Collosol Argentum may be applied every half hour with results which often surpass those obtainable by the use of stronger preparations. Issued in ½-oz. bottles with dropping pipette.

COLLOSOL **ARGENTUM** OPHTHALMIC

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## VITAMIN B-COMPLEX

**U. D.** Vitamin B-Complex is a sufficiently potent preparation containing the important factors—Thiamin, Pyridoxine, Pantothenic acid, Riboflavin, Niacinamide in liver extract base. It is of unsurpassed dependable purity and being in balanced proportions, allows a greater accuracy of dosage.

Packings

Boxes of 6 amp. of 1 & 2 c.c. & 10 c.c. R/C vials

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# *Bacillary* DYSENTERY

**High local concentrations can be safely maintained**

**'THALISTATYL' is practically non-toxic because it is only very slightly absorbed from the small intestine. High local concentrations can therefore safely be maintained. The bacteriostatic activity of 'Thalistatyl' is high and so the dosage is relatively small—7.5 g. initially with small maintenance doses.**

**'THALISTATYL' is indicated in the following conditions. Bacillary dysentery—Colitis—Infantile gastro-enteritis—Chronic amoebic dysentery (to clear up secondary infection of the ulcers).**

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## 'THALISTATYL'

TRADE MARK BRAND

PHTHALYLSULPHATHIAZOLE

**In 0.5 g. tablets in containers of 50, 100 and 500 tablets.**

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# remedies
## that speak of efficacy

**ASOKA CORDIAL CO.**
An unfailing uterine Tonic to correct intractable female diseases.

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A quick pick-me-up containing Glycerophosphates, Pepsin, Malt Extract and Alcohol in balanced proportions.

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**BROMO VALERIAN**
*COMPOUND*
A nervine Tonic—sedative and antispasmodic, indispensable in Hysteria.

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The Vitamin Laxative. Curative and Preventive. Containing vitamin B-Complex and Phenolpthalein.

# STREPTOPAS

Lepetit

**(Di-hydrostreptomycin tri-p-aminosalicylate)**

## A NEW ANTIBIOTIC

STREPTOPAS Lepetit is a neutral salt of di-hydrostreptomycin with p-aminosalicylic acid. Whilst containing 54% di-hydrostreptomycin base in the molecule, a bottle of 0.5 gm. STREPTOPAS Lepetit possesses a micro iological and therapeutic activity equal to 3 to 4 times that of the antibiotic in the form of an ordinary salt and at the same time considerably reduces the risk of developing Streptomycin-resistant strains of tubercle bacilli.

**ADVANTAGES :**

Possesses the properties of both the molecular groups (di-hydrostreptomycin and p-aminosalicylic acid) of which it is constituted.

* the bacteriostatic action on Mycobacterium T.
* the chemotherapeutic action in certain infections caused by Mycobacterium T.

Also several special advantages, including—

* potentialisation of activity with regard to di-hydrostreptomycin.
* practically neutral aqueous solutions can be administered, permitting the clinician to use the injection route he has previously chosen.
* low toxicity, enabling the clinician to reduce considerably the disadvantages associated with the antibiotic alone.
* high therapeutic activity—potentialisation effected by the use of the p-aminosalicylic constituent.

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**VIALS OF 0.5 GM.**

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*Indicated in:*

**Pre-and post-operative treatment**

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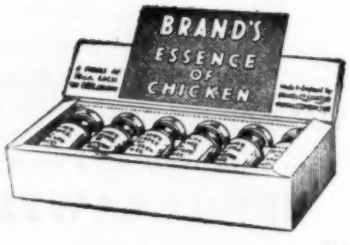
Clinical observation shows that these conditions are accompanied by protein depletion, resulting in a negative nitrogen balance. The condition of the patient may further aggravate this, owing to his inability to consume the food offered. In such cases care should be taken to include in the diet selected foods of high protein value which are palatable and easy to assimilate.

**Brand's Essence of Chicken is a first-class protein of animal origin. Being partly hydrolised, it is capable of easy ingestion, digestion and absorption. It is extremely palatable and may be taken either as a jelly or as a liquid. It is an ideal means of supporting convalescence and restoring a positive nitrogen balance.**

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IN 10 CC PHIALS

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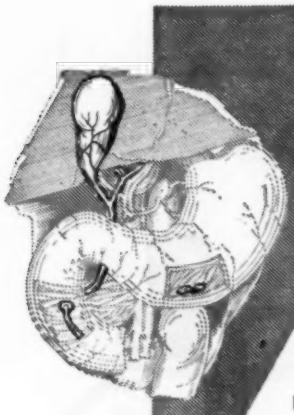
*Agents:* GRAHAMS TRADING CO., (INDIA) LTD.

CALCUTTA — MADRAS — BOMBAY — DELHI.



# BILE DEFICIENCY

HEXACHOL stimulates the activity of the liver, promotes the secretion and flow of bile. Has antiputrefactive and antiseptic action, establishes drainage of the biliary tract, aids emulsification and absorption of fats, prevents formation of cholesterol and biliary calculi, increases peristalsis of stomach and intestines.

HEXACHOL is beneficial in all cases of irritation, inflammation and infection of the biliary tract and in conditions of biliary insufficiency.

## HEXACHOL

*CIPLA*

COMPOSITION :

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|---|---|---|
| Cholic Acid | ... | 0.075 G. |
| Hexamine | ... | 0.225 G. |
| Inositol | ... | 5 mg. |

## NEOCHOLIN

*CIPLA*

EACH TABLET CONTAINS 0.25 G. DEHYDROCHOLIC ACID.

Cipla LABORATORIES BOMBAY-8.

LITERATURE SENT ON REQUEST.

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in

# B Vitamins

The earliest symptoms of Vitamin B deficiency are often difficult to detect without exhaustive and far-reaching tests. Fatigue, loss of weight, a finicky appetite, and frequent complaints of 'not feeling well' may be the first symptoms of Vitamin B deficiency. Whenever a dietary supplement is required to prevent the development of B avitaminosis, most doctors prefer WYETH'S 'PLEBEX', which contains the entire vitamin B complex as in yeast.

**'PLEBEX'** Elixir is available in bottles of 4 fluid oz.
**'PLEBEX'** Injections are available in 10 c.c. vials.

**"PLEBEX"**
TRADE MARK

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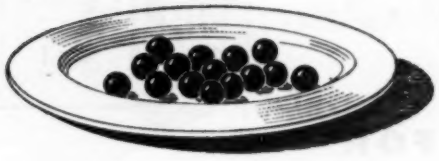
Stronachs

*Adexolin attacks the*

**BACKGROUND**

*of illness*



In strengthening the body's resistance to infection, the value of vitamins A and D is well established; but Adexolin—the Glaxo preparation of these vitamins in high concentration—is also a powerful aid to restoration of health after sickness, attacking the 'background of disease' through the natural mechanisms.

For your adult patients you can advise the popular protective routine—'one Adexolin capsule daily'; for the infant and child, Adexolin liquid is the preparation of choice, each drop supplying a balanced and standardised amount of the essential vitamins A and D.

GLAXO

ADEXOLIN

CAPSULES: Bottles of 25 and 100. Tins of 1,000.

LIQUID: Bottles of 14 cc. and 2 oz.

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Livules
PROTEOLYSED LIVER AND STOMACH POWDER WITH VITAMINS AND IRON
WITH FOLIC ACID
ALEMBIC CHEMICAL WORKS CO. LTD. BARODA

**FOR**

**ANAEMIAS**

ALEMBIC *Livules*

**(PLAIN OR WITH FOLIC ACID OR WITH $B_{12}$)**

*(EACH CAPSULE CONTAINS)*

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|---|---|
| Proteolysed Liver powder from | 50 grs. of Fresh Liver |
| Proteolysed Stomach ,, from | 15 grs. of Fresh Stomach |
| Ferrous Sulphate exc. | 1.5 grs. |
| Thiamine Hydrochloride ($B_1$) | 5 mgms. |
| Riboflavin ($B_2$) | 2 mgms. |
| Pyridoxine Hydrochloride ($B_6$) | 0.5 mgm. |
| Calcium Pantothenate | 1 mgm. |
| Niacinamide | 15 mgms. |
| Ascorbic Acid (C) | 30 mgms. |
| (Livules ç F. Acid) Folic Acid | 1.5 mgms. |
| (Livules ç B 12) Vitamin $B_{12}$ | 5 mcgs. |

Livules Plain:- BOTTLES OF 21, 42 & 84 CAPSULES.
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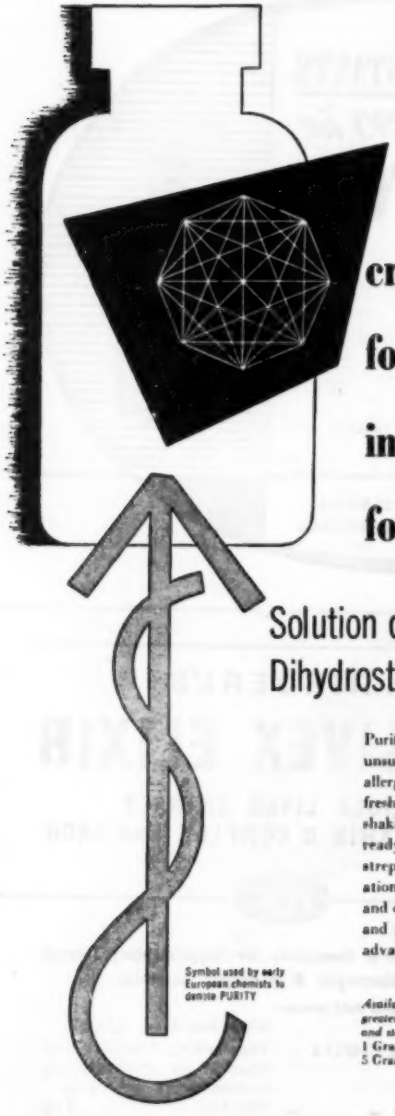

Symbol used by early European chemists to denote PURITY

**crystallized**

**for greater PURITY...**

**in solution**

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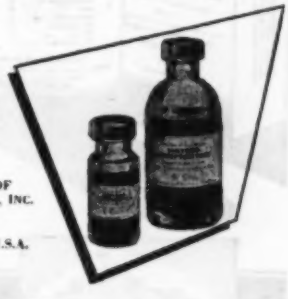

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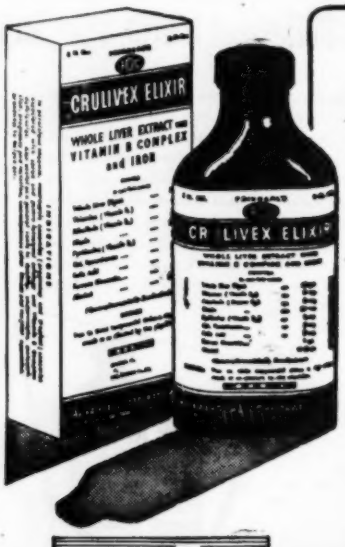


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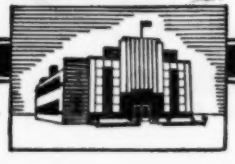

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VOL. XXI, No. 2 CALCUTTA NOVEMBER, 1951

*ORIGINAL ARTICLES*

## PLASMA ALKALINE PHOSPHATASE IN TOXAEMIA OF PREGNANCY*

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Since the first discovery of alkaline phosphatase in the plasma by Robison (1923), the role of this enzyme in the metabolism of bone has been studied by numerous workers. In pregnancy, the primary ossific centres of the fœtal bones commence to develop as early as the eighth week, but it is during the later part of gestation that fœtal bone formation becomes a rapid and active process. The investigations of Givens and Macy (1933), Mitchel (cited by Eden and Holland, 1948) and other observers point out the great fœtal demand for minerals, especially calcium and phosphorus, which occurs during the last trimester of gestation. It is only natural to assume that active fœtal bone formation should be associated with an increased demand for the enzyme essential for the process. Indeed, this is amply confirmed by the work of Meranze and co-workers (1937), Bodansky (1939), Young, King, Wood and Wootton (1946), and Hoch and Marrack (1948). All these investigators record a high level of phosphatase in the maternal plasma during the last three months of gestation.

In recent years estimation of the plasma alkaline phosphatase as a measure of hepatic efficiency has received considerable attention. Increase in its concentration has been observed in non-hæmolytic and obstructive jaundice by Roberts (1930, 1933), Bodansky and Jaffe (1931), Rothman, Meranze and Meranze (1936), and other investigators. Similar increase is stated to have been noted (cited by Rothman *et al.*, 1936) in malignant involvement of the liver also. Freeman, Chen and Ivy (1938) remarked that the plasma alkaline phosphatase is increased in all forms of liver injury, and its estimation is a more sensitive indicator of liver damage than a rise in serum bilirubin.

Several investigators (Barkeley and co-workers, 1924; Hofbauer, 1933; Herold, 1935; Rowe *et al.*, 1936; Dieckmann, 1941) believe that a state of hepatic dysfunction exists in toxæmias of pregnancy. Other observers (de Wesselow and Wyatt, 1924; Cruickshank, Hewitt and Couper, 1927) have not been able to convince themselves of this.

### MATERIALS AND METHOD

It has been pointed out that normal pregnancy causes an increase in the level of the plasma alkaline phosphatase. In order to evaluate the results obtained in toxæmias of pregnancy the normal value during pregnancy must be known. The difficulty in depending on the figures so far reported in the literature lies in the fact that not only the standard of unit of phosphatase used for estimations varies considerably with different authors, but also where the same standard has been employed the results do not wholly agree (Young *et al.*, 1946; Hoch and Marrack, 1948). Therefore, 93 cases of normal pregnancy were studied with a view to determine the normal standard of the plasma alkaline phosphatase. Among them, 78 were unselected cases from the antenatal clinic at varying periods of gestation. The remaining 15 cases were selected from the early months of pregnancy and were followed to term, an estimation being undertaken once every month. These 15 cases were also studied during labour and in early puerperium.

The group of toxæmias consisted of 146 cases. This was composed of 100 cases of pre-eclampsia, 18 cases of eclampsia, 10 cases of accidental hæmorrhage, 10 of essential hypertension and 8 of nephritis associated with pregnancy. The manner of selection of these cases has already been described (Mukherjee 1950).

In order to keep our results in conformity with those reported from the British Isles (Young *et al.*, 1946; Hoch and Marrack, 1948) the method employed was that of King and Armstrong (1934) as modified by King, Haselwood, Delory and Beall (1942).

### RESULTS

*Normal Pregnancy*—First Series. Of the 78 cases of normal pregnancy 39 belonged to the last trimester of gestation. The earliest pregnancy in our series was one of 9 weeks. The frequency distribution of the results obtained at different months of pregnancy are shown in Table 1. These figures indicate that up to the end of the second trimester of gestation the level

* This investigation was carried out at the research department of the Glasgow Royal Maternity and Women's Hospital with the aid of a scholarship granted to the author by the Government of India.

of the plasma alkaline phosphatase remains within normal limits. Statistical analysis, however, shows a significant tendency for the values to increase as gestation proceeds. The range of normality is exceeded in the eighth month of pregnancy, and during the following two months the plasma alkaline phosphatase rises to what may ordinarily be regarded as an abnormal level. This change is statistically significant. The shift to the right becomes evident about the middle of the second trimester and continues with increasing frequency as pregnancy advances. The average alkaline phosphatase content of the plasma in this series was $12{\cdot}24 \pm 1{\cdot}03$ during the last three months of pregnancy and $13{\cdot}65 \pm 0{\cdot}67$ at term.

TABLE 1—ALKALINE PHOSPHATASE IN NORMAL PREGNANCY. FREQUENCY DISTRIBUTION IN DIFFERENT MONTHS OF PREGNANCY

| Phosphatase units/100 ml. | Duration of Pregnancy—Lunar Months 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|
| 5·1—6 | 2 | 2 | — | — | — | — | — | — |
| 6·1—7 | 4 | 3 | 3 | 1 | — | — | — | — |
| 7·1—8 | 2 | 3 | 5 | 4 | 5 | — | — | — |
| 8·1—9 | — | — | — | 2 | 3 | — | — | — |
| 9·1—10 | — | — | — | — | — | 3 | — | — |
| 10·1—11 | — | — | — | — | — | 6 | 1 | — |
| 11·1—12 | — | — | — | — | — | 2 | 6 | 1 |
| 12·1—13 | — | — | — | — | — | 1 | 3 | 3 |
| 13·1—14 | — | — | — | — | — | — | 2 | 5 |
| 14·1—15 | — | — | — | — | — | — | — | 6 |
| No. of Cases | 8 | 8 | 8 | 7 | 8 | 12 | 12 | 15 |
| Average phosphatase content of plasma | 6·50 | 6·63 | 7·40 | 7·80 | 7·90 | 10·70 | 12·10 | 13·65 |
| | | | | | | | 12·24 | |
| Stand. Dev. | 0·71 | 0·72 | 0·64 | 0·65 | 0·55 | 0·94 | 0·94 | 1·00 |
| | | | | | | | 1·54 | |

Second Series. The results (Table 2) obtained in this series indicate the manner in which the alkaline phosphatase level varies in individual cases as gestation proceeds to term. In general they agree closely with those of the previous series (Fig. 1). The increase of the alkaline phosphatase content of the plasma during the last three months of pregnancy thus appears to be a consistent finding. According to the accepted normal standard (10 units per 100 ml., King, 1947), normal values were obtained in only 6 out of 45 tests done during the last trimester, an incidence of 86·7 per cent abnormality. These normal values were observed only before the thirty-second week of gestation. No patient during the last two months of pregnancy in either of these series of cases showed a "normal" level of plasma alkaline phosphatase. It is, however, interesting to note that the change which takes place at the end of the second trimester is sudden and abrupt.

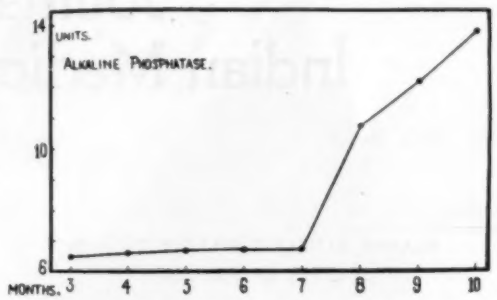


FIG. 1—Showing the change in the plasma alkaline phosphatase in different months of pregnancy.

The average alkaline phosphatase content of the plasma during the last three months of pregnancy, obtained from 84 tests done on 54 patients (both series taken together), is $12{\cdot}05 \pm 0{\cdot}92$ units per 100 ml. In the subsequent discussion on alkaline phosphatase in toxæmias of pregnancy this value will be considered as the normal standard.

TABLE 2—ALKALINE PHOSPHATASE IN NORMAL PREGNANCY, SHOWING THE RESULTS OF A FOLLOW-UP STUDY IN FIFTEEN CASES

| | *Lunar Months of Gestation* Phosphatase: Units per 100 ml. plasma | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| No. | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| 1 .. .. | 6·1 | 6·1 | 6·8 | 7·0 | 7·0 | 10·3 | 11·6 | 13·8 |
| 2 .. .. | — | 5·9 | 6·4 | 7·2 | 8·8 | 11·0 | 13·8 | 15·1 |
| 3 .. .. | 4·3 | 4·6 | 5·8 | 6·0 | 6·5 | 9·8 | 10·9 | 12·7 |
| 4 .. .. | — | 8·1 | 8·6 | 8·9 | 9·7 | 10·0 | 12·0 | 13·0 |
| 5 .. .. | 5·8 | 7·0 | 7·8 | 8·6 | 9·3 | 10·3 | 10·9 | 13·6 |
| 6 .. .. | 7·2 | 8·1 | 8·6 | 9·5 | 10·0 | 11·1 | 12·7 | 14·6 |
| 7 .. .. | 5·6 | 6·4 | 7·4 | 8·5 | 8·9 | 11·0 | 12·0 | 13·5 |
| 8 .. .. | 6·3 | 6·9 | 7·3 | 8·0 | 8·5 | 10·5 | 11·5 | 13·1 |
| 9 .. .. | 6·8 | 5·7 | 5·8 | 7·8 | 8·4 | 9·5 | 11·8 | 13·8 |
| 10 .. .. | 6·4 | 6·0 | 6·3 | 6·3 | 6·5 | 9·4 | 10·8 | 12·6 |
| 11 .. .. | — | 5·8 | 5·4 | 5·5 | 6·5 | 10·0 | 11·2 | 12·7 |
| 12 .. .. | 7·0 | 6·0 | 5·5 | 7·0 | 7·6 | 10·3 | 11·2 | 13·0 |
| 13 .. .. | — | 6·0 | 6·3 | 6·0 | 8·2 | 10·2 | 11·4 | 13·4 |
| 14 .. .. | 6·7 | 7·0 | 7·5 | 7·6 | 9·0 | 11·0 | 12·3 | 13·7 |
| 15 .. .. | 7·0 | 7·8 | 7·1 | 7·7 | 9·0 | 10·0 | 12·6 | 13·4 |
| Average .. | 5·8 | 6·5 | 7·2 | 7·5 | 8·2 | 10·3 | 11·8 | 13·5 |
| Stand. Dev. .. | 1·05 | 0·95 | 1·08 | 1·20 | 1·15 | 0·57 | 0·57 | 0·48 |
| Prob. Error. .. | 0·70 | 0·63 | 0·72 | 0·80 | 0·77 | 0·38 | 0·85 | 0·72 |

*Alkaline Phosphatase during Labour.*—The plasma alkaline phosphatase of the 15 cases belonging to the second series was estimated in the first and second stages of labour. Samples of blood were obtained when the first stage was established and again when the second stage was advanced. In 4 cases, labour was prolonged for more than 48 hours, and in these patients a third sample of blood was obtained when the first stage had lasted for nearly 40 hours. The results are shown in Table 3. For the purpose of comparison the values obtained at term are also included in this table. The interesting feature which is shown by all cases is the increase of the phosphatase content of the plasma during the second stage of labour. At the early stage of labour the phosphatase level does not show an appreciable difference from that at term.

TABLE 3—EFFECT OF LABOUR ON ALKALINE PHOSPHATASE IN NORMAL PREGNANT WOMEN. THE TABLE ALSO SHOWS THE VALUES OBTAINED IN EARLY PUERPERIUM

| | Alkaline Phosphatase: Units per 100 ml. plasma | | | | |
|---|---|---|---|---|---|
| *No.* | *At Term* | *First Stage* | *Late First Stage* | *Second Stage* | *Puerperium* |
| 1 .. | 13·8 | 14·1 | — | 16·3 | 9·2 |
| 2 .. | 15·1 | 15·0 | — | 19·0 | 11·4 |
| 3 .. | 12·7 | 12·5 | — | 15·1 | 8·6 |
| 4* .. | 13·0 | 13·5 | 16·3 | 20·6 | 12·0 |
| 5 .. | 13·6 | 13·5 | — | 16·5 | 10·3 |
| 6 .. | 14·6 | 14·9 | — | 16·9 | 9·5 |
| 7 .. | 13·5 | 14·2 | — | 17·0 | 9·0 |
| 8 .. | 13·1 | 13·0 | — | 16·8 | 11·6 |
| 9 .. | 13·8 | 13·8 | — | 17·8 | 10·4 |
| 10* .. | 12·6 | 13·3 | 17·0 | 21·2 | 15·8 |
| 11* .. | 12·7 | 13·0 | 18·6 | 21·9 | 13·9 |
| 12 .. | 13·0 | 13·6 | — | 17·0 | 10·0 |
| 13* .. | 13·4 | 13·0 | 18·0 | 22·0 | 16·4 |
| 14 .. | 13·7 | 14·1 | — | 18·2 | 12·7 |
| 15 .. | 13·4 | 13·9 | — | 17·0 | 10·8 |
| Average | 13·5 | 13·7 | 17·5 | 18·2 | 11·4 |
| Stand. Dev. .. | 0·72 | 0·74 | 1·11 | 2·53 | 2·45 |

* Prolonged labour: more than 48 hours.

When, however the labour is prolonged it increases during the late first stage. Compared with the values obtained at term, or in the early first stage of labour this increase is statistically significant. The effect of prolonged labour is further shown on careful analysis of the values obtained in the second stage. When labour was normal the average phosphatase level at this stage was $17{\cdot}05 \pm 0{\cdot}71$ units per 100 ml., which is almost equal to that found in the late first stage of prolonged labour. On the other hand, when labour lasted for more than 48 hours this average in the second stage was $21{\cdot}4 \pm 0{\cdot}45$ units per 100 ml. The difference between these two values indicates statistical significance. Compared with the basic level of alkaline phosphatase at term, the second stage was responsible for an increase of 26·3 per cent in normal labour, and 58·5 per cent in prolonged labour.

*Alkaline Phosphatase in Puerperium*—The plasma phosphatase on the fourth day of the puerperium was 11·4 1·64 units per 100 ml. (Table 3). The maximum and minimum values were 16·4 and 8·6 units respectively. When the cases of prolonged labour are excluded, the average alkaline phosphatase in the early puerperium appears to be $10{\cdot}3 \pm 0{\cdot}87$ units per 100 ml. The phosphatase content of the plasma in the puerperium is thus less than that at term. In the 4 cases of prolonged labour the puerperal level of phosphatase was $14{\cdot}5 \pm 1{\cdot}57$ units, which is not only higher than the accepted maximum normal limit for non-pregnant persons, but is also significantly higher than the values obtained at term.

*Pre-eclampsia*—The average alkaline phosphatase content of the plasma in pre-eclampsia was $16{\cdot}6 \pm 2{\cdot}30$ units per 100 ml. The co-efficient of variation is high, and is due to the wide scatter of the values obtained in individual cases. When the mild and severe pre-eclamptic patients are considered separately, it is found that the average for the former group is $14{\cdot}20 \pm 0{\cdot}71$ units and that for the latter, $18{\cdot}98 \pm 2{\cdot}65$ units per 100 ml. Compared with the value obtained in normal pregnancy these represent an increase of 17·9 and 57·5 per cent respectively. The progressive increase of the plasma alkaline phosphatase from normal pregnancy to mild and severe toxæmias show statistical significance at both stages. The frequency distribution of the values is shown in Table 4.

TABLE 4—FREQUENCY DISTRIBUTION OF ALKALINE PHOSPHATASE IN PRE-ECLAMPSIA

| *Units per 100 ml. Plasma* | *Mild Pre-eclampsia* | *Severe Pre-eclampsia* | *Total* |
|---|---|---|---|
| 12·1-14 .. | 22 | 3 | 25 |
| 14·1-16 .. | 25 | 10 | 35 |
| 16·1-18 .. | 3 | 9 | 12 |
| 18·1-20 .. | — | 12 | 12 |
| 20·1-22 .. | — | 7 | 7 |
| 22·1-24 .. | — | 5 | 5 |
| 24·1-26 .. | — | 1 | 1 |
| 26·1-28 .. | — | 1 | 1 |
| 28·1-30 .. | — | 2 | 2 |
| Total .. | 50 | 50 | 100 |
| Maximum .. | 16·5 | 29·5 | 29·5 |
| Minimum .. | 12·5 | 13·4 | 12·5 |
| Average .. | 14·26 | 18·98 | 16·60 |
| Stand. Dev. .. | 1·13 | 3·59 | 3·44 |
| Probable Error .. | 0·75 | 2·40 | 2·30 |

Comparison of the data presented here reveals that 28·6 per cent of the values in normal pregnancy, and 75 per cent of those in toxæmia exceeded 14 units per 100 ml. of plasma. Fifty-six per cent of mild pre-eclampsias, and 94 per cent of severe pre-eclampsias were above this limit. The maximum frequency in normal cases was between 10 to 12 units, in mild pre-eclamptics 14 to 16 units, and in severe pre-eclamptics 18 to 20 units per 100 ml.

It has already been pointed out that the plasma alkaline phosphatase increases progressively during the last three months of normal pregnancy. It was considered that this factor also required consideration in determining the nature and extent of the rise in phosphatase in pregnancy toxæmias. The results of an analysis of the values obtained in pre-eclampsia in relation to the duration of gestation are presented in

Table 5. It will be evident from the data presented here that the difference in the level of phosphatase which exists between normal, mild and severe eclampsia shows statistical significance even when the duration of pregnancy is taken into consideration.

*Alkaline Phosphatase and Diet in Toxæmias of Pregnancy*—Bodansky and Jaffe (*loc. cit.*) concluded from an experimental study that the physical and chemical processes associated with the state of nutrition are closely linked with the level of the alkaline

TABLE 5—ALKALINE PHOSPHATASE IN PRE-ECLAMPSIA, IN RELATION TO THE DURATION OF GESTATION

| *Duration of Gestation Weeks* | ALKALINE PHOSPHATASE: UNITS/100 ml. | | | | | |
|---|---|---|---|---|---|---|
| | *Mild Toxaemia* | | *Severe Toxæmia* | | *Normal** | |
| | Average | Stand. Dev. | Average | Stand. Dev. | Average | Stand. Dev. |
| 28—32 | | | | | | |
| Mild—9 | 13·1 | 0·91 | 16·2 | 1·01 | 10·7 | 0·94 |
| Severe—12 | (12·5—14·4) | | (13·0—14·4) | | (9·6—12·6) | |
| 32—36 | | | | | | |
| Mild—15 | 14·21 | 1·2 | 17·61 | 1·30 | 12·1 | 0·94 |
| Severe—8 | (13·6—15·7) | | (14·0—20·8) | | (10·8—13·4) | |
| 36—40 | | | | | | |
| Mild—26 | 14·88 | 1·31 | 20·13 | 3·22 | 13·65 | 1·00 |
| Severe—30 | (12·9—16·5) | | (18·0—29·5) | | (11·8—14·8) | |

* First series.

phosphatase in the plasma. Hough and Freeman (1942) demonstrated that the removal of protein from the diet causes an increase in the plasma phosphatase, and further observed that this change, which is of a reversible nature, appears soon after protein is restricted. Strauss (1938), Harden (1938) and several other investigators have suggested that a relationship exists between hypoproteinæmia and toxæmia of pregnancy. It was, therefore, considered that a study of the plasma alkaline phosphatase in relation to the protein content of the diet of the cases of pre-eclampsia might prove interesting. These cases were obtained from 3 units of the hospital. The total caloric value of the diet was the same in all cases, but the protein-content varied. The results are briefly presented below.

Unit A. Each patient received 102 gm. of protein daily, of which 71 gm. were first class protein. There were 42 cases in this group, 24 had a mild toxæmia, and 18 were severely toxæmic. The average alkaline phosphatase in this series was $15{\cdot}91 \pm 2{\cdot}17$ units, that for the mild cases was $14{\cdot}10 \pm 1{\cdot}24$, and for the severe cases, $18{\cdot}32 \pm 2{\cdot}05$.

Unit B. Thirty-three patients in this unit received 131 gm. protein daily, of which 115 gm. were first class protein. The average alkaline phosphatase in this series was $17{\cdot}29 \pm 3{\cdot}04$ units per 100 ml. The corresponding values for 15 cases of mild pre-eclampsia, and 18 cases of severe pre-eclampsia were $14{\cdot}27 \pm 0{\cdot}43$, and $19{\cdot}79 \pm 2{\cdot}24$ units respectively.

Unit C. The diet in this unit contained 75 gm. of protein, of which 43 gm. were first class protein. The average alkaline phosphatase in 25 patients in this series was $17{\cdot}03 \pm 2{\cdot}62$ units per 100 ml. In 11 cases of mild pre-eclampsia this was $14{\cdot}62 \pm 0{\cdot}60$ units, and the average in 14 cases of severe pre-eclampsia was $18{\cdot}92 \pm 3{\cdot}24$ units.

These figures show that the amount of protein intake has no noticeable effect on the alkaline phosphatase content of the plasma in toxæmias of pregnancy.

*Alkaline Phosphatase and the Clinical State of Toxæmia*—In order to determine whether the increase of the plasma phosphatase is directly related to the toxæmic process in pre-eclampsia, estimations were done at weekly intervals, and an attempt made to correlate the level of the plasma alkaline phosphatase with the clinical state of the patient. Forty-nine patients improved during their stay in the hospital, and in 51 cases the toxæmia either persisted or became worse. The results obtained in these two groups of cases are shown in Table 6. It will be seen that the level of the alkaline phosphatase is related to the manner of progress of the disease. With improvement in the clinical condition the level drops and nearly approaches the normal value at term. The decline is slow and gradual, and although this does not show statistical significance at every stage, it is associated with a progressive decrease of the co-efficient of variation. This indicates a significant shift of the values towards normal as the clinical condition improves.

The patients, where the condition deteriorated show a steady increase in the phosphatase level of the plasma. This is seen not only in the average, but also in the minimum and maximum values at each successive stage

TABLE 6—ALKALINE PHOSPHATASE IN RELATION TO THE CLINICAL COURSE OF TOXAEMIA IN PRE-ECLAMPSIA

| | *Improved—(49 cases)* | | | *Deteriorated*—(51 cases)* | | |
|---|---|---|---|---|---|---|
| *Weeks†* | Average | Stand. Dev. | Min.—Max. | Average | Stand. Dev. | Min.—Max. |
| 1 .. | 16·43 | 4·64 | 13·1—23·7 | 17·57 | 4·84 | 12·9—28·3 |
| 2 .. | 15·43 | 4·93 | 13·0—26·5 | 19·35 | 5·40 | 13·5—28·8 |
| 3 .. | 14·74 | 1·04 | 13·0—16·6 | 21·04 | 5·28 | 14·3—30·4 |
| 4 .. | 14·23 | 1·09 | 12·5—15·8 | 22·73 | 4·40 | 16·8—32·5 |

* Includes also cases where toxæmia was stationary.
† Refer to the period after admission into hospital.

of the investigation. The change in the plasma alkaline phosphatase caused by the aggravation of the toxæmia is statistically significant (Fig. 2).

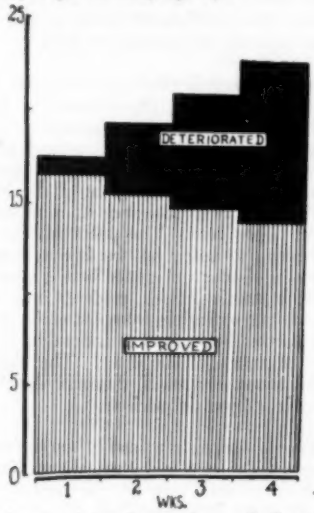


FIG. 2—Showing the change in the plasma alkaline phosphatase in pre-eclampsia as the clinical condition improves or deteriorates.

In a further study of the relation between the alkaline phosphatase and the clinical manifestations of toxæmia it was observed that neither albuminuria, nor œdema had any significant relationship with the level of the plasma alkaline phosphatase. Pre-eclamptic hypertension, however, appeared to have some deleterious effect on the phosphatase content of the plasma. The results were studied in relation to both systolic and diastolic blood pressures (Table 7). The correlation was more marked and significant with the diastolic pressure, especially when it was high (Fig. 3).

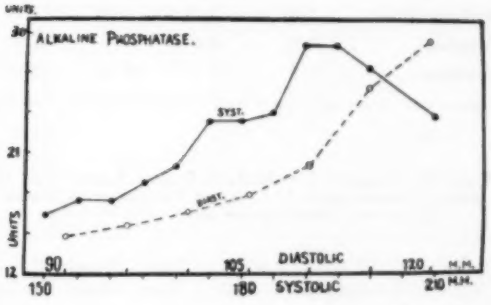


FIG. 3—Plasma alkaline phosphatase in pre-eclampsia, showing the changes in relation to blood-pressure.

TABLE 7—THE RELATION OF THE LEVEL OF ALKALINE PHOSPHATASE IN THE PLASMA IN PRE-ECLAMPSIA TO DIFFERENT LEVELS OF SYSTOLIC AND DIASTOLIC BLOOD PRESSURE

*Systolic Blood Pressure*

| *mm. Hg.* | *150* | *155* | *160* | *165* | *170* | *175* | *180* |
|---|---|---|---|---|---|---|---|
| Average .. | 16·53 | 17·68 | 17·62 | 18·82 | 20·22 | 23·71 | 23·81 |
| Stand. Dev. .. | 2·32 | 3·16 | 2·84 | 2·42 | 2·32 | 4·36 | 6·90 |
| Minimum .. | 13·2 | 13·7 | 13·8 | 14·5 | 16·5 | 16·7 | 13·5 |
| Maximum .. | 21·5 | 22·8 | 29·8 | 20·8 | 27·8 | 29·8 | 32·5 |

| *mm. Hg.* | *185* | *190* | *195* | *200* | *205* | *210* |
|---|---|---|---|---|---|---|
| Average .. .. .. | 24·0 | 29·06 | 29·0 | 27·32 | .. | 23·73 |
| Stand. Dev. .. .. | 4·3 | 2·9 | .. | 2·25 | .. | 7·23 |
| Minimum .. .. .. | 19·7 | 23·7 | .. | 23·2 | .. | 13·4 |
| Maximum .. .. .. | 28·3 | 32·5 | .. | 29·5 | .. | 29·8 |

*Diastolic Blood Pressure*

| *mm. Hg.* | *90* | *95* | *100* | *105* | *110* | *115* | *120* |
|---|---|---|---|---|---|---|---|
| Average .. | 14·93 | 15·70 | 16·41 | 17·73 | 20·03 | 25·96 | 28·96 |
| Stand. Dev. .. | 1·23 | 1·06 | 1·45 | 1·41 | 1·84 | 2·51 | 1·34 |
| Minimum .. | 12·8 | 14·4 | 13·4 | 16·7 | 18·6 | 21·2 | 25·6 |
| Maximum .. | 17·6 | 17·5 | 19·0 | 20·0 | 28·7 | 29·8 | 32·5 |

*Alkaline Phosphatase and Labour in Pre-eclampsia*—Toxæmia of pregnancy is known to predispose to obstetric shock. Frank, Seligman and Fine (1946) have demonstrated that the liver plays an important part in preventing the irreversible changes in shock. The present study was undertaken to determine if labour in toxæmia causes more metabolic upset and disturbance of the hepatic functions than that in normal pregnancy. Fifty-one cases of pre-eclampsia, where the toxæmia was persistent were studied for this purpose. These cases will be reviewed in two groups.

TABLE 8—ALKALINE PHOSPHATASE DURING LABOUR AND IN EARLY PUERPERIUM IN PRE-ECLAMPSIA. THE RESULTS WERE OBTAINED FROM 39 CASES OF NORMAL LABOUR

| | *Before Labour* | *First Stage* | *Second Stage* | *Puerperium* |
|---|---|---|---|---|
| Average .. | 20·71 | 21·03 | 26·29 | 16·74 |
| Stand. Dev. .. | 5·05 | 4·63 | 4·33 | 3·00 |
| Probable Error. .. | 3·38 | 3·10 | 2·90 | 2·01 |
| Maximum .. | 32·5 | 32·0 | 37·1 | 12·5 |
| Minimum .. | 15·5 | 15·3 | 21·4 | 20·6 |

TABLE 9—ALKALINE PHOSPHATASE DURING LABOUR AND IN EARLY PUERPERIUM IN 12 CASES OF PRE-ECLAMPSIA, WHERE LABOUR WAS PROLONGED FOR MORE THAN 48 HOURS

| No. | *Before Labour* | *Early First Stage* | *Late First Stage* | *Second Stage* | *Puerperium* |
|---|---|---|---|---|---|
| 1. D. .. | 23·2 | 23·0 | 31·3 | 39·8 | 28·5 |
| 2. McA. .. | 17·0 | 18·1 | 25·0 | 33·8 | 20·4 |
| 3. McD. .. | 20·0 | 20·0 | 25·9 | 36·2 | 26·6 |
| 4. G. .. | 22·7 | 22·0 | 28·3 | 36·8 | 24·5 |
| 5. P. .. | 28·6 | 29·2 | 34·5 | 40·8 | 31·3 |
| 6. O. .. | 29·1 | 30·0 | 37·1 | 46·2 | 29·0 |
| 7. C. .. | 14·9 | 15·4 | 23·1 | 31·8 | 20·0 |
| 8. L. .. | 17·9 | 19·0 | 25·0 | 34·9 | 22·2 |
| 9. F. .. | 22·6 | 22·0 | 28·8 | 39·1 | 27·5 |
| 10. McN. .. | 22·0 | 21·0 | 26·8 | 34·0 | 25·7 |
| 11. S. .. | 15·2 | 16·0 | 22·5 | 32·0 | 28·8 |
| 12. .. | 22·8 | 23·2 | 30·4 | 39·9 | 28·7 |
| Average .. | 21·33 | 21·57 | 28·22 | 37·11 | 26·10 |
| Stand. Dev. .. | 4·78 | 3·24 | 5·09 | 4·49 | 3·40 |
| Prob. Error. .. | 3·20 | 2·17 | 3·40 | 3·01 | 2·28 |

The first group consists of 39 cases, where the labour followed a normal course. Plasma phosphatase was estimated in these cases in the first stage and in the late second stage of labour. In the second group of 12 cases labour was prolonged for more than 48 hours. In these cases a further estimation was done when the first stage had lasted for more than 40 hours. The results are shown in Tables 8 and 9. The behaviour of the alkaline phosphatase in the first series shows a close resemblance to that in normal labour (Table 3). The pre-eclamptic patients had, indeed, a higher initial level of phosphatase in the plasma. The change observed at the onset of labour is insignificant. In the second stage the increase is sufficient to indicate statistical significance, and amounts to 26 per cent of the initial value. Normal labour, under similar conditions, was found to cause an increase of 26·3 per cent.

When the labour is prolonged in a case of toxæmia the increase in alkaline phosphatase is much more marked than that in normal pregnancy. As in normal labour the change in the value noticed in the early first stage is negligible, but the second stage caused the phosphatase to rise by 74 per cent of the initial value. Prolonged labour in normal pregnancy raised the value by only 58·5 per cent. The difference is significant. It is interesting to note that the late first stage values also showed a similar difference between normal pregnancy and toxæmia. It is evident from these data that in a patient suffering from toxæmia of pregnancy prolonged labour imposes a considerable strain on the functional efficiency of the liver. The significance of this in the management of dystocia developing in a pre-eclamptic subject is obvious.

*Alkaline Phosphatase and Puerperium in Pre-eclampsia*—In Tables 8 and 9 are shown the values obtained on the fourth day of the puerperium in the series of 51 cases just described.

Following a normal labour the alkaline phosphatase in the puerperium was $16{\cdot}74 \pm 2{\cdot}01$ units per 100 ml. This is 65 per cent higher than the corresponding value in normal pregnancy. The extent of the decrease of the plasma phosphatase in the puerperium, compared with the values obtained at term, amounts to 19·4 per cent in toxæmia and 23·2 per cent in normal cases.

When labour was prolonged the average plasma phosphatase on the fourth puerperal day was $26{\cdot}10 \pm 2{\cdot}28$ units per 100 ml. In spite of the slight decrease in value which occurred after child-birth, it still remained higher than the pre-parturition level of alkaline phosphatase in the plasma. This may be interpreted as a manifestation of a grossly impaired hepatic function. However, if the values obtained in the puerperium are compared with the maximum values observed during labour, it is seen that the rate of recovery after the confinement in pre-eclampsia is comparable with that in normal pregnancy. The fall in the alkaline phosphatase content of the plasma com-

pared with the value obtained in the second stage of labour amounted to—

1. When labour was normal,
   39·6 per cent in normal pregnancy, and
   36·3 per cent in pre-eclampsia.

2. When labour was prolonged,
   32·2 per cent in normal pregnancy, and
   29·7 per cent in pre-eclampsia.

It is evident that the changes in the liver caused by toxæmias of pregnancy are of a reversible nature.

*Eclampsia*—The average plasma alkaline phosphatase in 18 cases of eclampsia at the time of admission was 25·02 ± 2·26 units per 100 ml. The minimum and maximum values were 20·0 and 30·8 units per 100 ml. The average in eclampsia is 50·7 per cent higher than that in pre-eclampsia, and this difference is statistically significant. Three patients in this series were examined in the pre-eclamptic state as well. One to 3 days before the onset of convulsions the plasma phosphatase in these patients was 19·76 ± 0·81 units per 100 ml. During eclampsia the value increased to 24·86 ± 1·93 units. In 9 patients in this series the convulsive stage persisted for more than a day. On the first day the phosphatase level was 24·91 units ± 0·83, but on the following day it was 32·64 ± 1·97 units per 100 ml. This increase was shared by the maximum and minimum values alike, and indicates statistical significance. There were 3 deaths in this series. Twenty-four hours before the fatal outcome the plasma alkaline phosphatase in these patients was 29·8, 35·9 and 21·8 units. Six to 16 hours before death these values changed to 39·4, 39·5 and 39·7 units per 100 ml. respectively. When recovery occurred the phosphatase level of the plasma diminished progressively during the convalescence. By the second day it was 19·66 ± 2·17 units per 100 ml. End of the first week showed a further decrease, and by the twelfth day normal values were restored in all but 2 cases. All patients showed normal levels of plasma phosphatase by the end of the third week of convalescence.

TABLE 10—ALKALINE PHOSPHATASE IN ECLAMPSIA

| No. | Pre-convulsive | No. of Convulsions 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| 1. R. .. | 20·6 | 22·5 | 26·8 | .. | .. | .. | .. | .. | .. |
| 2. A. .. | 18·5 | 21·8 | .. | .. | .. | .. | .. | .. | .. |
| 3. H. .. | 20·2 | 20·8 | 21·0 | .. | 24·5 | .. | 26·0 | .. | 30·0 |
| 4. D. .. | .. | .. | 23·7 | 30·8 | 32·4 | 35·7 | .. | .. | .. |
| 5. McK. .. | .. | .. | .. | 18·6 | 19·5 | 20·0 | 23·4 | 29·8 | 34·0 |

An accurate study of the relationship between the alkaline phosphatase level and the number of convulsions was difficult, because of individual variation of the values in the pre-convulsive stage. However, the results obtained in 5 patients who were examined at frequent intervals and after each convulsion (3 of these cases were first seen in the pre-eclamptic stage) are shown in Table 10. It will be seen that the alkaline phosphatase content of the plasma increases with the number of convulsions (Fig. 4). Nevertheless, the number of fits does not seem to be the only factor responsible for this change. In one patient (R.) after 2 fits the phosphatase level rose by 6·2 units above the value in the pre-convulsive stage, but in another patient the same number of convulsions caused an increase of less than 1 unit per 100 ml. Again, between the second and the fourth convulsions the increase in phosphatase was by 8·7 units in one patient (D.), and 3·5 units in another (H.).

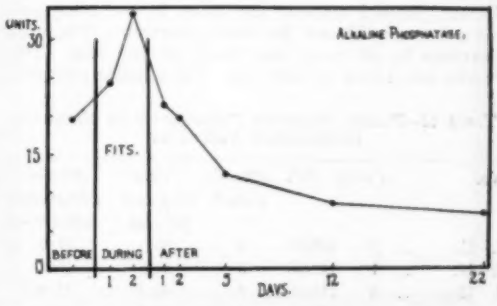


FIG. 4—Showing the effect of convulsion on the plasma alkaline phosphatase in eclampsia.

*Alkaline Phosphatase and Labour in Eclampsia*—In order to avoid the interfering influence of intrapartum convulsions the results obtained in only 5 cases in this series, where the last convulsion occurred 4 to 8 hours before the onset of labour have been considered

for this study. These are shown in Table 11. Unlike the two previous groups of cases, labour, even of normal

TABLE 11—PLASMA ALKALINE PHOSPHATASE DURING LABOUR IN ECLAMPTIC PATIENTS

| No.* | Alkaline Phosphatase units/100 ml. | | |
|---|---|---|---|
| | *Convulsive Stage* | *First Stage* | *Second Stage* |
| 1. Dm. (5) .. | 24·8 | 29·3 | 38·7 |
| 2. Dv. (4) .. | 30·8 | 32·5 | 41·6 |
| 3. R. (2) .. | 26·8 | 29·4 | 43·0 |
| 4. Ds. (7) .. | 35·7 | 36·7 | 45·0 |
| 5. McC. (4) .. | 28·8 | 29·6 | 44·5 |
| Average .. | 29·38 | 31·50 | 42·56 |
| Stand. Dev. .. | 2·69 | 2·58 | 2·58 |

* Figures in parentheses indicate the number of convulsions.

duration, following soon after an eclamptic fit is accompanied by a marked increase in the phosphatase level of the plasma. This becomes evident even in the early first stage of labour. Compared with the value obtained in the convulsive stage, this increase, although it amounts to only 2·12 units in this series, is statistically significant. The change caused by the second stage is, however, considerable. The net increase varied from 9·3 (Ds.) to 16·2 (R.) units, with an average of 13·18 ± 2·18 units per 100 ml. This corresponds to a gain of 44·5 per cent of the antepartum value obtained in the convulsive stage. Thus, it appears that the increased metabolic strain caused by labour of even normal duration immediately following an eclamptic state interferes with the hepatic functions much more than labour in pre-eclampsia. The importance of this lies in the obstetric management of a case of eclampsia.

*Nephritis Complicating Pregnancy*—There were 8 cases in this series. All had a history of renal disease, showed albumin and casts in the urine and were between 32 and 36 weeks pregnant. The urea clearance in all cases was below 60 per cent. The results are shown in Table 12. The alkaline phosphatase of the plasma in this series was, average 11·51 ± 0·88, minimum 9·8, and maximum 13·8 units per 100 ml. These values are not significantly different from those observed in normal pregnancy. Neither the level of blood pressure nor the extent of renal damage, as measured by albuminuria and urea clearance, seemed to affect the phosphatase level in pregnancy in a nephritic patient.

TABLE 12—PLASMA ALKALINE PHOSPHATASE IN NEPHRITIS COMPLICATING PREGNANCY

| *No.* | *Parity* | *B.P.* | *Albumin Esbach* | *Urea Clearance per cent.* | *Alkaline Phosphatase unit/100 ml.* |
|---|---|---|---|---|---|
| 1. C. .. | 2 | 160/94 | 9 | 60 | 11·0 |
| 2. M. .. | 1 | 145/85 | 3 | 54·5 | 10·7 |
| 3. K. .. | 3 | 175/100 | 8 | 46·2 | 13·8 |
| 4. W. .. | 2 | 138/85 | 4 | 50·5 | 9·8 |
| 5. McQ. .. | 4 | 160/100 | 2 | 52·3 | 11·5 |
| 6. S. .. | 1 | 148/92 | 3 | 55·8 | 13·0 |
| 7. H .. | 2 | 150/90 | 2 | 49·7 | 11·8 |
| 8. B. .. | 2 | 160/95 | 4 | 58·4 | 12·1 |
| | | | | Average .. | 11·51 |
| | | | | S.D. .. | 1·34 |

*Essential Hypertension and Pregnancy*—There were 10 cases in this series. All were known to have hypertension before or during the first three months of gestation. No patient had an abnormal urea clearance. The duration of pregnancy was between 32 and 36 weeks. The results are shown in Table 13. The

TABLE 13—PLASMA ALKALINE PHOSPHATASE IN ESSENTIAL HYPERTENSION COMPLICATING PREGNANCY

| *No.* | *Parity* | *B.P.* | *Albumin Esbach* | *Urea Clearance per cent* | *Alkaline Phosphatase unit/100 ml.* |
|---|---|---|---|---|---|
| 1. S. .. | 1 | 200/130 | 3 | 94·5 | 14·2 |
| 2. P. .. | 2 | 180/120 | 2 | 113·0 | 11·6 |
| 3. B. .. | 2 | 210/135 | 6 | 91·6 | 16·5 |
| 4. McD. .. | 1 | 195/120 | 1 | 99·5 | 11·0 |
| 5. McK. .. | 1 | 190/115 | ± | 104·5 | 10·7 |
| 6. K. .. | 1 | 185/110 | — | 100·5 | 12·0 |
| 7. H. .. | 2 | 190/120 | — | 97·6 | 9·3 |
| 8. Pr. .. | 1 | 200/120 | 1·5 | 93·5 | 10·6 |
| 9. J. .. | 1 | 200/110 | 1 | 102·8 | 10·4 |
| 10. C. .. | 3 | 188/115 | 4 | 109·4 | 14·5 |
| | | | | Average .. | 12·07 |
| | | | | S.D. .. | 2·30 |

average alkaline phosphatase in this series was 12·07 ± 1·54 units per 100 ml. This is not significantly different from the value obtained in normal pregnancy. It is, however, interesting to note that in 3 patients where the hypertension was accompanied by marked albuminuria (S., B., & C.) the level of the plasma alkaline phosphatase was high. This albuminuria could not have been due to organic renal involvement, as the results of urea clearance will show. It seems that some cases of essential hypertension during pregnancy are associated with functional disturbances similar to those seen in pre-eclampsia.

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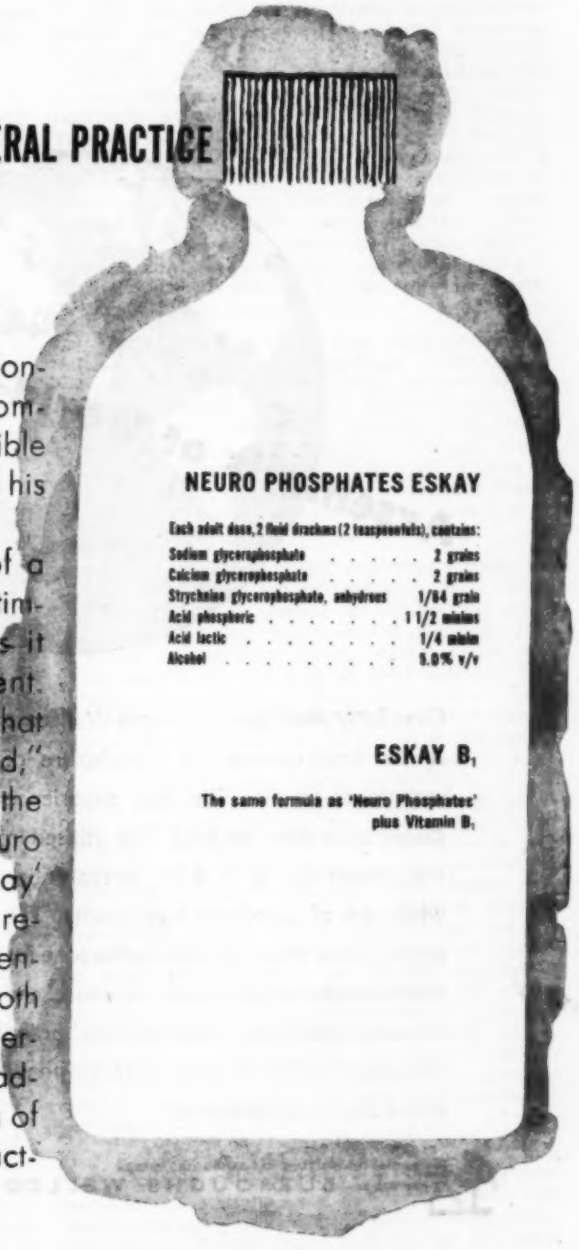
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*Accidental Hæmorrhage*—The data derived from 10 cases in this series are presented in Table 14. In 4

TABLE 14—PLASMA ALKALINE PHOSPHATASE IN ACCIDENTAL HAEMORRHAGE

| *No.* | *Before Hæmorrhage** | | *During Hæmorrhage* | *Shock* |
|---|---|---|---|---|
| 1. W. | .. | .. | 19·6 | + |
| 2. M. | .. | .. | 33·2 | ++ |
| 3. D. | .. | 13·9 | 29·5 | ++ |
| 4. P. | .. | .. | 16·8 | ± |
| 5. B. | .. | .. | 15·9 | 0 |
| 6. McI. | .. | .. | 17·3 | + |
| 7. McV. | .. | .. | 17·0 | + |
| 8. O'R. | .. | 15·1 | 15·5 | ± |
| 9. McK. | .. | 14·7 | 27·4 | ++ |
| 10. McG. | .. | 16·3 | 16·5 | 0 |
| Average | .. | 15·0 | 20·87 | |
| Stand. Dev. | .. | 1·08 | 6·05 | |

* Only values obtained within 72 hours before the onset of hæmorrhage are included.

patients the level of plasma phosphatase less than 72 hours before the onset of retro-placental hæmorrhage was known. The average alkaline phosphatase content of the plasma in accidental hæmorrhage was $20{\cdot}57 \pm 4{\cdot}05$ units per 100 ml. Individual values provide a more interesting study. Four patients in this series developed little or no shock as a result of the hæmorrhage. The average in these cases, 16·17 units per 100 ml., is not appreciably different from the pre-hæmorrhagic level of phosphatase in the whole series. The alkaline phosphatase in 3 patients, who were slightly shocked was 17·96 units. This is slightly higher than the pre-hæmorrhagic value. In 3 cases shock was severe, and their plasma phosphatase content was 30·0 units per 100 ml. This is considerably higher than the level of phosphatase before the onset of hæmorrhage and almost approaches that of eclampsia in its most severe form.

## DISCUSSION

All studies on plasma alkaline phosphatase in normal pregnancy indicate that during the first six months of gestation the values obtained are not different from those in normal non-pregnant persons. Abnormal values first appear in the seventh month of pregnancy. During the following weeks the increase in the plasma phosphatase is progressive. This has been clearly shown in the present analysis, and also in those reported by Cayla and Fabre (1935), Meranze *et al.* (1937) Bodansky (1939), Vermehren (1939) and Hoch and Marrack (1948). The actual values stated by these authors are not suitable for comparison because of the difference in the units employed for the estimation of the phosphatase. In a series of 220 cases belonging to the last 8 weeks of pregnancy Young, King, Wood and Wootton (1946) found 10·4 units of alkaline phosphatase per 100 ml. of plasma. Hoch and Marrack (1948) who employed the same unit of measurement found 11·3 units of phosphatase per 100 ml. between 27 weeks of pregnancy and term. These values agree closely with the results of the present investigation.

The cause of this sudden increase of alkaline phosphatase has, however, inspired several interesting hypotheses. Thus, Meranze and co-workers (1937) state that a "pathologic or a potentially pathologic state of bone metabolism in the mother or fœtus" is probably responsible for the high phosphatase level. Ramsay, Thierens and Magee (1938) observe that the high plasma phosphatase is likely to be associated with a disordered calcium and phosphorus metabolism in pregnancy. Bodansky (1939) suggests an increased parathyroid function as the cause. Young *et al.* (1946) point out that the high maternal phosphatase could be associated with infantile rickets and deficient calcium metabolism in the fœtus. The data on which these theories are based are inadequate.

Ebbs and Scott (1940) found high phosphatase values in twin pregnancy. Meranze, Meranze and Rothman (1937) also made a similar observation and mentioned a case of triplet where the phosphatase level was even higher. Our findings are not in accordance with this, but rather agree with those of Young, King, Wood and Wootton (1946). There were 4 cases of twin pregnancy in our series. The average which was 12·84 units is not appreciably different from that in single pregnancy.

Givens and Macy (1933) and other workers on maternal metabolism during pregnancy have shown that there is a great demand for calcium and phosphorus during the last three months of gestation. This is related, among other things, to the deposit and formation of osseous tissue in the fœtal skeleton. The phosphatase content of the fœtal blood is low. Estimation in the cord blood of 21 normal new-born babies gave values from 3·0 to 6·1 units, with an average of $4{\cdot}67 \pm 0{\cdot}81$ units per 100 ml. Considering the state of fœtal metabolism the plasma concentration of the enzyme essential for osteogenesis is extremely poor. It is reasonable to conclude that under such circumstances a compensatory mechanism develops in the mother whereby the requirements of the fœtus can be met as and when necessary. A low phosphatase content of the fœtal blood has been observed also by Stearns and Warweg (1933), and Meranze and co-workers (1937). Examination of the placental tissue for histochemical reactions for alkaline phosphatase shows that there is an appreciable difference in the phosphatase content of the chorion between early and late months of pregnancy (Figs. 5 and 6, *vide* Plate). These findings suggest that the high level of maternal phosphatase is a physiological response to the demands made by the fœtus during the last three months of gestation.

The behaviour of the plasma alkaline phosphatase in toxæmias of pregnancy does not appear to be connected with this physiological process. Reference has already been made to the work of various investigators, who consider that pregnancy toxæmias are associated with some degree of hepatic dysfunction. The increase in plasma phosphatase from 12·24 units in normal pregnancy to 18·98 units in severe toxæmia, and 25·02 units in eclampsia is significant. That this increase

is related to the toxæmic process is further shown by the change caused by the alteration of the clinical condition of the patient. In this connection, it is interesting to observe that neither nephritis, nor essential hypertension complicating pregnancy gives rise to any significant variation from the values obtained in normal pregnancy, unless the hypertension is unbalanced (Mukherjee and Govan, 1950) and associated with marked albuminuria. Thus, determination of the plasma alkaline phosphatase as an aid to diagnosis in a composite group of toxæmias is not without limitations. In this respect the bilirubin excretion test appears to be more satisfactory. However, in determining the clinical course and progress of the disease estimation of the phosphatase seems to be of considerable value.

Increase of plasma phosphatase in accidental hæmorrhage is related to the degree of shock which is present. Shock is attended with a fall of blood pressure, diminished oxygenating capacity of the blood and vascular spasm. These factors, superimposed on a pre-existing vascular spasm caused by toxæmia, considerably undermines the functional capacity of the liver cells. The histological changes in the liver in shock described by Davidson and co-workers (1946), and the experimental observation made by Engel, Harrison and Long (1944) on the effects of anoxæmia on the liver cells offer support to the suggestion made above.

Sufficient attention has not been paid to the effect of labour on alkaline phosphatase. Meranze *et al.* (1937) mention that they observed an increase in the phosphatase level of the plasma during "active labour." The present investigation shows that this increase is related to the duration of labour. When labour is prolonged the plasma phosphatase rises to an abnormally high level. Labour is attended with prolonged muscular contraction and increase of intra-abdominal pressure. The strain imposed on the metabolic functions of the liver by these factors evidently calls upon the reserve capacity of the organ and explains the increase of the plasma phosphatase during labour. When labour is prolonged in a case of toxæmia the significance of this finding is greatly increased. The vivi-perfusion experiments of Frank, Seligman and Fine (1946) demonstrate the importance of a normally functioning liver in preventing the irreversible changes in shock. The results of the present investigation show that the hepatic functions suffer appreciably in toxæmias of pregnancy and considerably more so when the strain of a prolonged labour is superimposed on severe toxæmia.

## Summary

1. Study of the plasma alkaline phosphatase in 93 cases of normal pregnancy indicates that during the last three months of pregnancy the phosphatase values are high according to the accepted standards. It is suggested that this abnormal level of the plasma phosphatase is caused by the physiological demands made by the fœtus.

2. Plasma phosphatase in pre-eclampsia and eclampsia is abnormally high. This is believed to be due to hepatic dysfunction which accompanies toxæmias of pregnancy. The plasma phosphatase is high in accidental hæmorrhage associated with shock. Nephritis and compensated essential hypertension in pregnant women do not raise the phosphatase level of the plasma.

3. Labour increases the phosphatase level of the plasma. This is marked in prolonged labour and in presence of toxæmia. During the first week of the puerperium, however, the values start to return to normal. The rate of decline to some extent depends on the height attained by the phosphatase during labour.

## References

Barkeley, C., Dodds, E. C. and Walker, A. L.—*J. Obst. & Gynæc. Brit. Emp.*, **31**:20, 1924.
Bodansky, M.—*Am. J. Clin. Path.*, **9**:36, 1939.
Bodansky, A. and Jaffe, H. L.—*Proc. Soc. Exper. Biol. Med.*, **29**:199, 1931.
Cayla, J. and Fabre, F.—*Compt. rend. Soc. Biol.*, **120**:748, 1935.
Cruickshank, J. N., Hewitt, J. and Couper, K. L.—Medical Research Council, Sp. Rep. No. 117.
Davidson, L. S., Lewiss, J. H., Jagnon, H. J. and Adams, M. A.—*New England J. Med.*, **234**:279, 1946.
Dieckmann, W. J.—Toxaemias of Pregnancy, Henry Kimpton, London, 1941.
Ebbs, J. H. and Scott, W. A.—*Am. J. Obst. & Gynec.* **39**:1043, 1940.
Eden, T. W. and Holland, E.—Manual of Obstetrics, Churchill, London, 1948.
Engel, F. L., Harrison, A. M. and Long, C. N. H.—*J. Exper. Med.*, **79**:9, 1944.
Frank, H. A., Seligman, A. M. and Fine, J.—*J. Clin. Invest.*, **25**:22, 1946.
Freeman, S., Chen, Y. P., and Ivy, A. C.—*J. Biol. Chem.*, **124**:79, 1938.
Givens, M. H. and Macy, I. G.—*Ibid*, **120**:7, 1933.
Harden, B.—Discussion on Dodge, E. F. and Frost, T. F. in *J. A. M. A.*, **111**:1898, 1938.
Herold, K.—*Zntralbl. f. Gynak.*, **159**:166, 1935.
Hough, V. H. and Freeman, S.—*Am. J. Physiol.*, **138**:184, 1942.
Hoch, H. and Marrack, J. R.—*J. Obst. & Gynæc. Brit. Emp.*, **55**:1, 1948.
Hofbauer, J.—*Zentralbl. f. Gynak.*, **157**:35, 1933.
King, E. J. and Amstrong, A. R.—*Canad. M. A. J.*, **31**:376, 1934.
King, E. J., Haselwood, G. A. D., Delory, G. E. and Baell, D.—*Lancet*, **1**:207, 1942.
King, E. J.—Micro-Analysis in Medical Biochemistry, Churchill, London, 1947.
Meranze, T., Meranze, D. R. and Rothman, M. M.—*Am. J. Obst. & Gynec.*, **33**:444, 1937.
Mukherjee, C. and Govan, A. D. T.—*J. Obst. Gynæc. Brit. Emp.*, **57**:94, 1950.
*Idem*—*Glasgow M. J.*, **31**:77, 1950.
Robison, R.—*Biochem. J.*, **17**:286, 1923.
Ramsay, J., Thierns, V. T. and Magee, H. E.—*Brit. M. J.*, **1**:1199, 1938.
Roberts, W .M.—*Brit. J. Exper. Path.*, **11**:90, 1930.
*Idem*—*Brit. M. J.*, **1**:734, 1933.
Robinson, R.—*Biochem. J.*, **17**:286, 1923.
Rothman, M. M., Meranze, D. R. and Meranze, T.—*Am. J. M. Sc.*, **192**:526, 1936.
Rowe, A. W., McMannus, M. A. and Plummer, A. J.—*Am. J. Obst. & Gynec.*, **31**:856, 1936.
Stearns, G., and Warweg, E.—*J. Biol. Chem.*, **102**:749, 1936.
Strauss, M. B.—*Am. J. M. Sc.*, **195**:723, 1938.
Vermehren, E.—*Acta. med. scandinav.*, **100**:254, 1939.
de Wesselow, O. L. V. and Wyatt, J. M.—Modern Views on Toxæmias of Pregnancy, Constable, London, 1924.
Young, J., King, E. J., Wood, E. and Wootton, I. D. P.—*J. Obst. & Gynæc. Brit. Emp.*, **53**:251, 1946.

# HYPERTENSION: WITH REFERENCE TO ITS SURGICAL TREATMENT

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## Introduction

Hypertension presents one of the most difficult medical problems of the present day. It is not easy to realize its ravages because of its insiduous onset, absence of early incapacitating symptoms and unwarranted optimism of the patients and their relatives. No other disease exacts such an appalling toll of human efficiency and society leadership because it primarily affects the intelligentsia of society. Nearly all cases of so-called cerebral accidents which include both intracranial hæmorrhages and cerebrovascular thrombosis, most cases of heart disease, including coronary thrombosis and the great majority of Bright's disease with renal insufficiency have hypertension as a common factor.

The terms essential or benign hypertension are both misnomers because it has been proved recently that it is not essential to have an increased blood pressure to force blood through an inadequate arterial system, nor is the disease ever benign although serious manifestations may not appear for many years.

The blood pressure may be temporarily or continuously elevated in a wide variety of conditions; for example, in cerebral trauma the blood pressure may rise very rapidly with the increase of intracranial pressure either from hæmorrhage or œdema. Certain tumours, such as basophilic adenomas of the pituitary and certain adrenal tumours, can produce a continuous or recurrent elevation of blood pressure as can chronic nephritis.

The highly complex disease syndrome of hypertension may be characterised at first by only a mild elevation of blood pressure and without any symptoms, but its manifestations may be many with great variations in progress. Headache, nervousness, insomnia, easy fatigability are common complaints. Others may come to notice with the beginning of cardiac or renal insufficiency. Yet others are first manifested by a stroke, either a central hæmorrhage or cerebral thrombosis. The rapidity of progress varies as much as its manifestations. A few may live out a normal life span without complaints or accidents, but the majority sooner or later show its results. No one can prognosticate what may happen to any individual case. The most common course is for the disease to progress slowly but steadily. These characteristics of the disease make the evaluation of any treatment extremely difficult.

## Relation of Hypertension and Kidney Pathology

Goldblatt *et al.* (1934) showed conclusively that renal hæmodynamic state, produced a continuous elevation of blood pressure without any evidence of impaired renal function in the beginning. It has been shown by many that the interference with the blood supply to the kidneys produces an excessive amount of renal pseudoglobulin called "rennin" which is liberated into the renal vein. This rennin is thought to react with another pseudoglobulin, probably arising in the liver, and named "preangiotonin"—that renin activator of Page (1939)—forms an active vasopressor substance called "angiotonin". This angiotonin causes arteriolar constriction and raises the peripheral vascular resistance, with a consequent elevation in blood pressure, but it is not easy to answer the question of what causes the changes in hæmodynamics which eventually produce the angiotonin.

Damage to the renal parenchyma alone will not produce hypertension. It has been shown that both the kidneys can be surgically removed in animals without the production of hypertension. But if the blood supply to the kidneys are interfered with, continued elevated blood pressures are seen. Crile and DeCourcy operated upon the adrenal with excision of one gland or a portion of each and also they denervated the adrenal. After extensive experience they were convinced that extirpation of adrenal tissue was of no benefit.

## Medical Treatment

Although medical treatment is usually inadequate certain general measures are indicated. Reduction in weight is important in all overweight patients. Sufficient rest at night and, if possible, a rest period after the noon meal, are valuable. Worry is a recognized factor but to eliminate worry is easier said than done. Dietary regulations have helped in many cases. In Western countries the rice diet has gained much popularity and has often lowered the pressure, but this is of little value in our country. The salt-free diet, or in other words, low sodium diet, has proved of great value in some cases. Potassium thiocyanate has been recommended by certain clinicians, but it has to be given continuously and with controlled blood levels. In all these types of treatment often there is a return of high blood pressure during or after completion of the treatment.

## Operative Procedure

It has been demonstrated experimentally by Trueta, Prichard and others that stimulation of the splanchnic nerves upset the hæmodynamics of the kidney and produced elevated blood pressure. Therefore, treatment of hypertension is directed towards eliminating factors which upset the hæmodynamics of the kidneys.

Removal of the splanchnic nerves and the lower thoracic and the upper lumbar sympathetic chain showed great benefit. Although this operation was not done originally for hypertension, its benefit in lowering the blood pressure was learnt when this was done for other conditions.

Surgery is the best form of therapy for certain selected cases, sympathectomy being carried out to reduce the formation of angiotonin by preventing increased action of the sympathetic nervous system which disturbs the hæmodynamics of the kidneys.

For the purpose of evaluation of treatment, selection of cases for suitable therapy and to prognosticate the result of various methods of treatment a classification of these hypertension cases seems essential. All hypertensive cases can be placed in one of the four groups:—

Group 1—Those with systolic blood pressures above 160 mm. Hg. and diastolic above 100 mm, but without symptoms. Fundus changes are nil or very minimal—Grade 1 of Wagener and Keith (1939). They show no evidence of cardiac, renal or cerebral involvement. As a rule they show a good response to sodium amytal, sodium nitrate or cold pressor tests, thereby indicating that the pressure is labile.

Group 2—Those with symptoms of hypertension but without any symptoms and signs of decompensation. Heart and kidney involvement may be present in varying degrees, but they are not the presenting symptom complex. They need not show any cerebral impairment although they complain of its symptoms. The retinal changes may be of Grade 2 with changes in the A.V. ratio and fullness of the veins and with occasional slight exudates, but no swelling or failing vision. The blood pressures are not as constantly labile as in Group 1.

Group 3—Those who may have had some decompensations. Cerebrovascular accidents, cardiac or renal insufficiency may have been noted but has responded to medical management. There may be considerable deficiency of cardiac and renal function; fundus examination may reveal hypertensive retinopathy; and they may not show much variation of blood pressure to sodium amytal, sodium nitrate or cold pressor tests.

Group 4—This is characterised by papillœdema. They also show exudates and hæmorrhages and other findings of hypertensive retinopathy on fundus examination.

The above-mentioned grouping of cases are really arbitrary and are based upon the severity of the disease, the appearance of the fundi and the progress of the disease.

Age is an important factor. The younger group have a much higher percentage of good results than the older age group whatever may be the form of treatment. Every case is given general medical care and treatment for a period of at least two to three months and if satisfactory results are not obtained, surgery is contemplated. Too often the physicians watch the progress of the disease until irreparable damage has resulted, either in the brain, heart or kidneys and then consider surgical treatment. This is neither fair to the patient nor to the surgical treatment.

We believe that any patient showing progressive hypertension should have the benefit of surgical consideration. It is not considered advisable to operate on people above 50 years of age or with sufficient renal damage as to increase the blood urea above 40 mgm. per cent and nonprotein nitrogen above 50 mgm. per cent. Similarly, we do not operate on people who are in cardiac failure and who have had a cerebrovascular accident within the previous three months.

Craig (1948) of the Mayo Clinic reports that patients without any treatment or with medical treatment alone, over a period of five years, have a mortality rate of 25 per cent for those in group 1. Group 2 shows a rate of 45 per cent; group 3, a rate of 80 per cent and group 4, 100 per cent.

Group 1 usually are the younger age patients. They do well with medical or surgical treatment. No doubt some cases get excellent results with medical management and treatment, whereas others often show only temporary lowering of the blood pressure. Even without any treatment some few cases live out a normal span of life without symptoms. Therefore, this group of patients should be treated surgically only when they show progression of the process.

Group 2 cases often need surgical treatment. They are all given a period of good medical care preoperatively and in a few instances surgery may not be necessary. Too much time should not be spent watching for further progression of the disease lest the heart or kidneys decompensate.

Group 3 includes the bad risk cases but many excellent results have been obtained by surgery. Peet emphasises that surgery is the only hope for increasing the life expectancy in these cases.

Since almost all cases of group 4, where they have paillœdema, die within one or two years they are operated upon regardless of the extreme poor risk, except that the requisites of a blood urea below 40 mgm. per cent and age below 50 are still adhered to.

Robert, Sterling and Palmer, in their comparative study on medical and surgical treatment of hypertension, observe that the younger patients of group 1 get 70 per cent excellent results by surgery as compared to 20 per cent by medical management. Surgical treatment was more effective in lowering the blood pressure even if only temporarily. This response was much recorded in group 1, moderate in groups 2 and 4, but not so impressive in group 3 cases. (In the majority of the group 3 series, 75 per cent, the blood pressure returns to hypertension levels, even though less than previous levels in 3 to 5 years). In their summary they emphasise that twice as many good results are obtained by surgery as by medical management and also that sympathectomy is to be regarded as the treatment of choice for hypertension with papillœdema.

It is to be considered that sympathectomy is a palliative treatment and not a curative procedure. It should be advised only after the patient has first failed to respond to medical management with rigid restriction of sodium ion in the food and limitation of general activities.

The surgical treatment also varies considerably in the extent of the sympathetic denervation. The first approach consisted of rhizotomy and splanchnic resections, which was suggested in 1923 by Danielpolo. In 1930 Peri first performed the operation by the supradiaphragmatic approach and in 1933 Peet by the bilateral sub-diaphragmatic technique. Smithwick in 1938 did a supradiaphragmatic sympathectomy from T10 to L2 splitting the diaphragm and in 1942 from T4 to L2. Poppen revised this technique to supra- and infradiaphragmatic without splitting the diaphragm. Even though these are the major changes in the technique of sympathectomy there had been many who have used other variations.

The technique we use in our clinic is a combination of Smithwick's and Poppen's. We do a bilateral operation in two stages of infra- and supradiaphragmatic extent by the extraperitoneal and extrapleural approach. Portions of 11th and 7th ribs are resected and the sympathetic chain from T4 to L2 with the three splanchnic nerves are removed in one piece. This technique gives the maximum benefit of sympathetic surgery to these patients. It is expected that these extensive sympathectomies will give better and longer lasting results compared with other less extension procedures.

### References


Craig, W. M.—*The Interne*, March, 1948.

Foa, P. P., Woods, W. W., Peet, M. M. and Foa, N. L.—*Arch. Int. Med.*, **71**:357-369, 1943.

Goldblatt, H., Lynch, T., Hanzal, R. F., and Sommerville, W. W.—*J. Exper. Med.*, **59**:347-79, 1934.

Grimson, K. S.—*Ann. Surg.*, **114**:753-75, 1941.

Keith, N. M., Wagener, H. P. and Barker, N. W.—*Am. J. M. Sc.*, **197**:332, 1939.

Levy, R. L., White, P. D., Stroud, W. D. and Hillman, C. C.—*J. A. M. A.*, **128**:1059, 1945.

Page, I. H.—*J. Indiana M. A.*, **32**:562, 1939.

Peet, Max. M. and Isberg, Emil M.—*Ibid*, **130**:467-72, 1946.

Peet, Max. M., Isberg, Emil, M. and Basset, Robert C.—*Surg. Gynec. & Obstet.*, **86**:673-79, 1948.

Peet, Max. M., Woods, W. W. and Braden, S.—*J. A. M. A.*, **115**:1875, 1940.

Smithwick, R. H.—*Surgery*, **7**:1-8, 1940.

*Idem—Arch. Surg.*, **49**:180, 1944.


## *PRACTITIONERS' CORNER*

## PERIARTHRITIS SHOULDER


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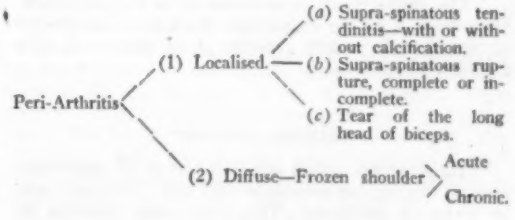
Painful shoulder accounts for a considerable proportion of our minor orthopædic out-patients. But apart from its frequency, it is an interesting subject for study. Obscure in its etiology, uncertain in its prognosis, and unsettled in its treatment, it forms a definite clinical entity. To classify all the causes of shoulder pain is not within the scope of this paper, referring as it does exclusively to that clinical syndrome variously called, periarthritis, frozen shoulder, lesions of the musculo-tendinous cuff and so on.

### Etiology and Pathology

The disease is common in adult males seldom occurring under the age of 20. The right shoulder is much more often affected than the left. Bilateral affections are distinctly rare. As a rule no other joints are affected.

In the study of this disease it is rather unfortunate that much more attention is paid to the supraspinatus tendon and muscle. The recent literature is more devoted to this muscle and its tendon and the term periarthritis has fallen into disrepute. It is more logical, however, that the whole musculo-tendinous cuff of the shoulder joint consisting of the supraspinatus, infra-spinatus and teres minor and the long head of the biceps muscles and their tendons and the intervening fibrous aponeurosis together with the subacromial bursa, should be viewed upon as one working unit. Codman claims "that complete rupture of the supra-spinatus is the most common cause of prolonged disability from industrial accidents to shoulder and incomplete ruptures account for the majority of minor shoulder disabilities." To claim so much for this mere strip of a muscle is perhaps too much if we consider that this muscle is after all only a part of the general musculo-tendinous cuff. Any inflammatory or degenerative or even traumatic affection is likely to affect more or less the entire cuff rather than a part of it, though very often the brunt borne by the supraspinatus part of it is clinically the most arresting. Believing that a lesion of the musculo-tendinous cuff may manifest itself clinically either as a localised affection or as a diffuse affection the whole disease group may be reclassified as follows:—



When the lesion is relatively localised it is often the supraspinatus muscle that is affected as it forms the most vulnerable part of the cuff, by virtue of its anatomical position. When the lesion is more diffuse the condition is loosely spoken of as frozen shoulder. If we accept this classification, the problem of etiology and pathogenesis becomes clearer.

Codman first thought that all these conditions are different manifestations of subacromial bursitis but later on he corrected himself and said that the bursitis is only secondary to peri arthritis or arthritis. A diagnosis of bursitis is as sound as a diagnosis of peritonitis without any reference to the primary cause. But the onset of bursitis has a great clinical importance because it is the one factor that causes the acute onset of disease or its acute exacerbations. In fact a calcified tendon may be actually symptomless until it causes a reactionary bursitis.

What is the nature of this peri-arthritis? Is it a true inflammation or is it a degenerative process? There are several factors that play a role in the causation of this condition.

1. *Infection*—A majority of patients show evidence of some active focal sepsis; and not a few give a history of gonorrhœa. There is usually a low grade fever and slight leucocytosis in the acute phase of the disease. There is some malaise and constitutional upset, factors which point to a mild infective process at work.

2. *Trauma and degenerative processes*—The infective factor is not by itself powerful enough and even if it is so, there is no reason why it should choose the shoulder joint for its victims. Obviously there is a local predisposing factor. The musculo-tendinous cuff is placed between the joint capsule on the one side and the bony arch formed by the acromion and coracoid on the other. The result is that whenever the shoulder is abducted, the cuff is ironed out and rolled on the acromio-coracoid arch;—a factor leading to repeated trauma of a mild nature. The gravity of this repeated trauma will be more significant if the patient follows an occupation which involves a repeated movement of abduction and external rotation of the shoulder joint. The repeated trauma leads to a degenerative process and deficient vascularity and when a traumatic factor supervenes a tear occurs readily.

3. *Endocrine factor*—Some claim an endocrine dysfunction as a causative factor also.

The pathogenesis of calcification is also important. Some view it as the expression of an abortive attempt at repair while others consider it as the result of a degenerative process.

## Clinical Picture

Those points which are common to all manifestations of the disease are considered first. The onset may be acute or insidious. The acute onset denotes invariably that the sub-acromial bursa is involved and the acute phase clears up as the bursitis subsides.

*History of injury*—It is absent in the majority of cases except in frank ruptures of the tendon.

*Pain*—Though the lesion is in the musculo-tendinous cuff, yet it is curious that the worst pain is felt at the site of insertion of the deltoid muscle. This is due to two factors: the spasm of the deltoid and also referred pain along the circumflex nerve. The pain may be very severe causing acute misery or may only be a dull ache. It often radiates down the arm and is often dubbed as a neuralgia, or it may shoot up the neck. The pain is worse in the night. There are no sensory disturbances, nor are the nerve trunks tender on palpation. Codman emphasises the markedly deleterious effect that the condition has on the general and mental state of those who suffer from it. "In addition to the pain and sleepless nights, anxiety that it may not be possible to work again, and the lack of help from his doctor, the fact that he may be accused of hysteria or malingering may at length cause the sufferer to lose his self respect and brooding over hard-luck, to take to drink."

*Cases of acute onset*—Patients are ill, run a low moderate leucocytosis.

*Localised forms—(a) Supra-spinatus tendinitis*—Apart from the pain which is already described, the patient has a definite tender spot just below the acromion, *i.e.,* directly over the supra-spinatus. The movements of the shoulders are not limited but the patient complains of pain while abducting, the pain starting not at the onset but as abduction has progressed up to 60° and the pain persists till the range of abduction has reached 120° when the pain disappears. The pain is felt during that range of abduction when the supra-spinatus is impinging over the acromion process. This painful arc is very characteristic.

*(b) Rupture of the supra-spinatus tendon*—Complete ruptures are rare and occur only after violent trauma, usually a dislocation. Supra-spinatus initiates abduction and so the characteristic feature of this condition is the failure of initiating abduction in spite of a contracting deltoid muscle.

*(c) Calcification of the supra-spinatus tendon*—has not much clinical significance and the symptoms are just the same as in tendinitis. But when the calcification is massive, the bulk of the tendon is greater and so the friction between it and the acromion is greater and symptoms, therefore, are more marked.

*Diffuse form*—Is somewhat more common, at least in our hospital practice. Most often the onset is acute and severe. The patient is very apprehensive of any movement but on persuasion can attain full flexion and extension without any pain but abduction and external rotation are painful. There is no particular tendor spot.

### Diagnosis

Before one ventures on a diagnosis of periarthritis, tuberculous disease of the shoulder should be ruled out. The fundamental sign of arthritis is pain and limitation of all the movements of the joint. It is a safe rule in orthopædics that if in any joint all movements are limited and painful to any degree the diagnosis of an arthritis is most likely, and if even one movement is full and painless, the diagnosis of arthritis is most unlikely.

There is one important sign named after Daqbarn which is present in all cases where the tenderness is over the greater tuberosity. It consists in the disappearance of the tender point on full abduction due to the fact that the painful part has disappeared underneath the acromion.

General periarthritis is to be distinguished from supra-spinatus lesions by the absence of a tender spot and of the characteristic painful arc of 60-120° abduction.

X-ray pictures should be taken in all cases unless the diagnosis is beyond all doubt. The skiagram may show calcification, or a narrowing of the space between the humeral head and the acromion, or a normal picture or rarely, to our painful surprise, caries sicca.

### Prognosis

Prognosis is good with proper and early treatment. Some cases run a chronic course and result in adhesions which require a manipulation under an anæsthetic. Generalised periarthitis runs a definite course and this course seems to be independent of the type of treatment given. Sometimes it disappears as suddenly as it came on. Usually it disappears in two or three weeks.

*Supra-spinatus tendinitis*—Where a definite tender spot is present, excellent results are obtained by injection of novocaine. 10 to 20 c.c. of 1 per cent novocaine is injected into the tender spot. The needle must be inserted boldly and pushed in until the tender spot is reached when the patient will complain of a severe pain of the same type as he is suffering from. Injection of the drug must result in immediate relief of pain and the patient should be able to swing his shoulder round freely. Such a successful injection invariably results in cure. But the patient must be warned that a few hours after the injection the pain will come back in an exaggerated intensity but in a day or two the pain will gradually disappear not to recur again.

Cases with *limited calcification* will yield to the same treatment. But in such cases diathermy is of great use as it helps to improve circulation and thus absorption of the calcification. In obstinate cases the simpler procedure is to wash off the deposit by saline by means of two wide-bore needles inserted into the mass under fluoroscopic control, or alternatively an open operation is done.

The treatment of *actual rupture* is again either by an open operation and suture or by prolonged immobilisation of the shoulder in abduction by means of a plaster spica.

Cases of *diffuse periarthritis* offer a far more formidable problem from the treatment point of view. In the acute stage the patient is put to bed and attention is paid to any focal sepsis. The patient is put on plenty of fluids and nourishing food. Locally sometimes fomentations are not tolerated by the patient as it aggravates the pain. Cold packs may then be applied. The shoulder is splinted in the most comfortable position, but as soon as possible, active momements should be encouraged or else adhesions form and lead to a stiff joint. For pain a sedative should be given. The usual A.P.C. powder is as good as any. So long as there is no tender spot, novocaine injection is useless. Diathermy is of some use but no immediate results can be expected. The worst form of treatment in the acute stage is manipulation but in late cases where the movements are limited due to organised adhesions manipulation under an anæsthetic is carried out.

In short, apart from general measures and control of pain, local treatment resolves itself into hot or cold packs or diathermy, early active movements, and encouragement to the patient.

### References


1. Burns, B. H., and Ellis, V. H.—Recent Advances in Orthopædic Surgery., J. & A. Churchill Ltd., 1937.
2. Dickson, J. A., and Crosby, E. H.—*J. A. M., A.*, **99**:2252, 1932.
3. Mayer,—*J. Bone & Joint Surg.*, **19**:640, 1937.
4. Philip, D. Wilson—*Brit. M. J.*, **2**:1261, 1939.
5. Watson Jones, R.—Fractures and Joint Injuries., E. & S. Ligingstone, 1943.


## ABDOMINAL TUBERCULOSIS

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*Poona.*

In India, Abdominal Tuberculosis is quite a common disease. Why it is so is quite obvious. Our poverty, our lack of medical aid, our poor diet, our unhygienic conditions, all contribute to the spread of this disease. It appears to me that this disease was known to the ancients as part of the description of abdominal trouble given by Hippocrates resembles very closely the present day description of abdominal Tuberculosis. From my experience of the last twenty years it seems to me that the disease has been on the increase during the last ten years or so, in my district. I am sorry I cannot support my statement with the necessary data as they are not available. Knowing very well how badly our hospital records are maintained, this difficulty of obtaining accurate statistics will be understood by you all. Yet, I will try my best to furnish you with the same, wherever possible.

In discussing abdominal tuberculosis I shall restrict myself to the following four types:

(*i*) Tuberculosis of the small intestine or ulcerative tuberculosis of the small bowel.

(*ii*) Tuberculosis of the ileocæcal junction or hypertrophic tuberculosis of the cæcum.

(*iii*) Tuberculosis of the mesenteric glands.

(*iv*) Tuberculosis of the peritoneum.

*Tuberculosis of the small intestine* (ulcerative tuberculosis of ileum)—It is mostly the lower end of the ileum that gets often affected in this variety. Sometimes the disease may spread to the upper regions of the intestine. It may also be associated with tuberculosis of the mesenteric glands or of the ileocæcal junction. The tubercle bacilli settle in the Peyer's patches of the ileum where they produce one or many tuberculous ulcers. These ulcers spread round the gut along the lymphatics and during their healing, form tuberculous stricture, which may be single or multiple. From the Peyer's patches the disease may spread to the outer coats of the small intestine and affect the peritoneum. In countries where milk is taken without being boiled, the disease may occur in the primary form. In British Isles, where cattle are frequently affected with tuberculosis and where milk is taken unboiled, this primary form is mostly seen in children. It is caused by bovine type of tubercle bacillus. In most other countries including India this type is uncommon. In India, this is due to the rarity of tuberculosis in cattle and secondly to the habit of the people of not drinking unboiled milk. This primary type reacts well to anti-tuberculosis measures.

The secondary type is common among young adults, suffering from pulmonary tuberculosis. It is said that 95 per cent of the people showing tuberculous cavities in the lung suffer from ulcerative tuberculosis of the small intestine. This type is due to the swallowing of the sputum by the tuberculous patient. It is produced by human type of bacilli. Extrapulmonary tuberculous lesions may cause ulcerative tuberculosis of the small bowel, the bacilli reaching the intestine *via* blood. This, however, is of rare occurrence. Ulcerative tuberculosis does not produce any marked symptoms excepting attacks of colicky pain in the abdomen, diarrhœa and loss of weight. These symptoms are, however, commonly seen in phthisical patients even without bowel involvement. The disease may remain symptomless but rarely remains so when the ulcers are in the acute stage. As such the disease may remain unsuspected for a long time. In the acute stage there is a marked tenderness all over the abdomen accompanied by severe pain and high temperature. The mere finding of the tubercle bacilli in stools is not diagnostic of the disease, as tubercle bacilli can be found in stools of pthisical patients without any bowel involvement. Presence of occult blood and R.B.C. in stools suggest ulceration of the bowel but not definitely of tuberculous origin. If, however, a tuberculous patient does not show any improvement even after the lung lesion has come under control, ulcerative tuberculosis of the small bowel should strongly be suspected and even more so if he is suffering from colicky pain in the abdomen and diarrhœa alternating with constipation. In addition to this, the presence of R.B.C., occult blood and tubercle bacilli in stools makes the bowel lesion almost certain. Associated with the above symptoms there may be rise in temperature. The colicky pain is due to muscular spasm. The diarrhœa may be fatty. In that case it is due to lymphatic obstruction produced by tuberculous mesenteric glands. Non-fatty diarrhœa is possibly due to marked hyperperistalsis.

In a large percentage of cases of pulmonary tuberculosis, where abdominal symptoms are absent, a correct diagnosis of ulcerative tuberculosis of the bowel can be made on the strength of x-ray findings. It has been stated by *Brown and Sampson* that in a case of lung tuberculosis if repeated barium meal examinations of the gastro-intestinal tract show stasis in the stomach and small intestine while the meal passes very rapidly through the cæcum, ascending colon and hepatic flexure with spastic defects in them, a diagnosis of ulcerative tuberculosis of small intestine can definitely be made. In those cases which have developed tuberculous strictures the x-ray will reveal dilated loops of intestine, filled with fluid and gas. The presence of calcified gland shadows in the x-ray plate supports the diagnosis of ulcerative tuberculosis of the small intestine if other symptoms mentioned above are also present.

Ulcerative tuberculosis of the small bowel is liable to certain complications, *e.g.*—tuberculous strictures, which later on, may give rise to subacute or acute intestinal obstruction. Such an obstruction may also result from adhesions produced between loops of intestines by the extension of the disease to the peritoneal coat. Extension of the disease may also occur into the greater omentum which forms a transverse rolled up mass in the upper part of the abdomen. Sometimes the extension may result in a cold abscess which may burst outside near about the umbilicus and give rise to a tuberculous sinus. If a communication is established with the intestine a fæcal fistula may result. Perforation of a tuberculous ulcer is very rare.

In treating these cases, the lung lesion must firstly be brought under control by proper medical and surgical treatment. Patients suffering from intestinal tuberculosis should be kept in fresh air. They require good, nourishing, high protein, low residue diet. The diet should be supplemented by a liberal quota of vitamins specially vitamins B and C. Those suffering from diarrhœa should be treated with astringent drugs such as kaolin, opium, codein, bismuth, etc. For colicky pain preparations containing belladonna or hyoscyamus are found useful. At present many of these cases can be treated with streptomycin injections with extremely good results. The usual dose recommended by Mason and his colleagues is half a gram twice a day continued for a fortnight. Most of their cases were doing well even six months after the treatment. Patients deve-

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loping intestinal obstruction need immediate operation. In such cases, the diseased-portion of the gut may be resected, followed by an end to end anastomosis, or a simple short-circuiting operation may be carried out. If because of extreme distension of the intestine any anastomotic procedure is considered to be dangerous then an extraperitoneal resection of the diseased intestine may prove a safe procedure.

*Tuberculosis of ileocæcal region or hypertrophic tuberculosis of the cæcum*—This disease is usually limited to the ileocæcal region but may spread proximally to the ileum or distally to the colon. The neighbouring lymph glands are usually enlarged. The disease may be associated with ulcerative tuberculosis of the small intestine. Many people, including Sir Arthur Hurst, think this disease to be uncommon. It is said that many cases recorded as ileocæcal tuberculosis are, in fact, cases of regional ileitis (Crohn's disease). I personally do not think this disease to be so rare. I have myself operated on, as many as thirty eight cases of ileocæcal tuberculosis during the last twenty years, thirty five of them occurring in females between the age 20 to 30 years. Why the disease affects females in such a large number remains unexplained. This disease is mostly a primary affection, very few occurring secondary to pulmonary tuberculosis.

The disease starts insidiously the patients' only complaint being loss of weight, loss of appetite and some discomfort in the right iliac fossa. As the disease progresses, the patient starts complaining of colicky pain in the abdomen, some hours after meals. This pain may occasionally be accompanied by nausea or vomiting. By this time a hard lump in the right iliac fossa may be apparent to the patient or to the physician. The lump is usually mobile. These attacks of pain with a lump in the abdomen may be mistaken for recurrent appendicitis. At a later stage patient shows typical symptoms of sub-acute or chronic intestinal obstruction. The lump gets more or less fixed in the right iliac fassa by this time. The small intestine now shows visible peristalsis with a ladder pattern on the abdominal wall which is associated with loud borborygmi. On auscultation of the abdomen a marked hyperperistaltic note is heard which may have a metallic ring, suggesting a definite dilatation of the intestine. The patient at this stage looks dehydrated and anæmic. Rarely does this condition pass into an acute intestinal obstruction. At this stage, the disease resembles closely to cancer of the cæcum. Cancer, however, occurs at a later age and shows marked cachexia. Diarrhœa alternating with constipation is more common in cancer cases. A filling defect distal to the cæcum after a barium enema may suggest cancer of the cæcum. It is very difficult to differentiate ileocæcal tuberculosis from regional ileitis (Crohn's disease). If, however, a barium meal shows a filling defect in the distal part of the ileum in the form of a string (Kanton's sign) this goes in favour of regional ileitis. There are other conditions, like actinomycosis of the cæcum, lymphosarcoma, hodgkin's disease, etc., which resemble ileocæcal tuberculosis and may have to be differentiated from it.

In the early stage of the disease, a pure medical line of treatment should be followed, as in ulcerative tuberculosis of the ilium. But in cases where definite signs of intestinal obstruction are present the treatment is surgical. Some surgeons, because of high mortality following after a radical resection of the diseased part have advocated a short-circuiting operation. I however favour radical resection. I have done twenty-six radical resections with only two deaths. I advocate this, provided the following conditions are present: *viz.*, (1) the patient must be afebrile at least for a fortnight before the operation, (2) his anæmia must be corrected by proper treatment including transfusion of blood, (3) he should put on weight while under observation, (4) the sedimentation rate of his red cells should be within normal limits. At present the above four requisites can be sooner brought about by the use of streptomycin. Streptomycin is also very useful for any flare-up that may occur after a radical resection. For those, who are not accustomed to intestinal surgery I would advise them to carry out this radical resection extraperitoneally.

*Tuberculous Enlargement of the Mesenteric Glands*—Tuberculous enlargement of the mesenteric glands often occurs secondarily to tuberculosis of ileocæcal junction (hypertrophic tuberculosis of the cæcum) or ulcerative tuberculosis of the small intestine. Then its signs and symptoms correspond closely to the primary disease mentioned above. The glands, however may get affected primarily without any obvious lesion in the intestine. In some cases the mesenteric glands are affected along with the cervical, axillary, groin, or thoracic lymph glands. The affection in such cases is considered to be blood-borne.

The primary tuberculous affection of the mesenteric glands is seen in children and in young adults between the age of 18 to 35 years. In children, this disease gives rise to severe abdominal pain with intestinal distension. Diarrhœa is often present. There is rise in temperature with marked wasting. Such a condition is called tabes mesenterica. On examination these glands may be felt abdominally or per rectum. The mass of glands is often situated in the right iliac fossa. In less severe cases, the glands may give rise to attacks of abdominal pain with vomiting. These attacks may be mistaken for acute appendicitis.

In adults, the glands in the early stage may give rise to vague abdominal symptoms such as pain, flatulence, fullness of the stomach etc. There is usually some loss of weight. Such a case may resemble any one of the following: *viz.*, (1) chronic appendicitis (2) chronic cholecystitis (3) peptic ulcer (4) chronic amœbiasis of the colon. If by careful examination one can eliminate all the above four conditions then one can reasonably diagnose the case as suffering from early enlargement of the mesenteric glands. Many of these prove to be due to tuberculosis. During the last five years I have operated on 129 cases of chronic abdominal pain, out of which more than 52 per cent prove to be cases of enlargement of mesenteric gland. 95 per cent of these were proved in the followup to be

due to tuberculosis. If along with the above symptoms a lump is felt in the abdomen which moves horizontally but not vertically then one can suspect the case to be one of tuberculous mesenteric glands. The other signs which will confirm the diagnosis are: (1) an increased sedimentation rate (2) the presence of calcified gland shadows in the x'ray plate and (3) rise in temperature. The lump produced by the enlarged mesenteric glands may resemble hodgkin's disease, lymphosarcoma or a mesenteric cyst.

Tuberculosis of the mesenteric glands may produce certain complications. The glands may press on the duodenum and cause its obstruction. If the glands caseate and form a cold abscess this may burst into the peritoneal cavity and give rise to tuberculous peritonitis. When the glands are situated in the mesentery of the sigmoid colon, they may give rise to mesenteric irritation and produce megacolon. The glands may get adherent to the intestine and cause a kinking, thus giving rise to intestinal obstruction. During my practice I have operated on nine such cases.

Tuberculous mesenteric glands in children should be treated medically. Streptomycin has been found of immense value in these cases. I have personally observed big glandular masses completely subsiding with streptomycin treatment. Early cases in adults, with chronic abdominal pain should in my opinion be explored and if no other disease is found the appendix removed. I believe the removal of the appendix proves beneficial to the patient. These cases however need prolonged medical treatment and in some cases the use of streptomycin may bring about a rapid improvement.

*Tuberculous peritonitis*—This affection is noticed in children and in young adults. In Western countries it occurs more in children than in adults while my experience so far as my district is concerned is just the reverse;—young adults being more affected than children. I believe the same is true of other extrapulmonary tuberculous diseases, *viz*, Pott's disease and tuberculosis of the bones and joints.

Tuberculous peritonitis may take the acute or the chronic form. The acute form, when it is associated with generalised miliary tuberculosis, rarely shows any sign or symptom as these are completely masked by those of generalised miliary tuberculosis. When however acute tuberculous peritonitis occurs alone, in its more severe stage, it may resemble perforative peritonitis, acute appendicitis or acute intestinal obstruction from any of which it may be very difficult to differentiate. An exploratory laparotomy only revealing the true nature of the condition. In some of these cases urine examination may show the presence of tubercle bacillus. In its less severe form it may resemble typhoid fever. There is usually a high rise of temperature, and enlarged spleen and leucopenia with relative lymphocytosis. From this, it can, however, be differentiated by the negative Widal test and a negative blood culture. This form is usually caused by extension of the infection from the neighbouring tuberculous lesion, such as tuberculosis of the intestine, of the mesenteric glands, of the uterine tubes, ovaries etc. In young females the disease is usually due to an extension of the infection from the uterine tubes and ovaries, amenorrhœa being then a prominent symptom. Prognosis of this form was usually bad, most of the cases proving fatal in two to three month's time. The use of sterptomycin, however, has brought about a distinct change for the better.

The chronic form usually begins very insidiously, the patient complaining of loss of appetite, a feeling of tiredness and general malaise. Later on he finds his abdomen gradually getting bigger. He suffers now from abdominal pain. There may be occasional diarrhœa. Flatulence may be a troublesome symptom. Temperature may go up to 102° or 103°F. However the disease often remains afebrile. There is gradual loss of weight.

Clinically the ascitic type of chronic peritonitis occurs in two varieties: (1) generalised, (2) localised. The generalised variety may be recognised by the uniform abdominal distension and the shifting dullness (which may ocasionally be absent owing to the contraction of the mesentery produced by the tuberculous process). The skin over the abdomen gets stretched and looks shiny. The umbilicus starts getting everted and becomes slit like. The superficial veins get dilated. In the male child a congenital hydrocele may develop if the patent processus vaginalis gets filled with the fluid. In children below the age of five, the preascitic stage may be attended with marked flatulence and, therefore, be confused with rickets, Hirschsprung's disease or cœliac disease. A positive Mantoux test before the age of five will go in favour of tuberculous peritonitis. Rickets can be differentiated by the bony changes which are apparent in the x-ray plate. Hirschsprung's disease can be detected by the barium enema. The generalised variety may also resemble ascites produced by cardiac or renal disease, by cirrhosis of the liver or by secondary carcinomatous affection of the peritoneum. From these it may, however, be differentiated by the specific gravity of its fluid which is 1018, by the marked increase of lymphocytes in its fluid and by the guinea-pig inoculation test. After removal of the ascitic fluid by tapping one may be able to palpate the rolled-up omentum or the enlarged mesteric glands.

The localised variety may resemble an ovarian cyst, a retroperitoneal tumour or a polycystic kidney. An ovarian tumour however is dull on percussion, while the localised variety is usually resonant. The x-ray plate taken after a pneumoperitoneum may differentiate the localised variety from the retroperitoneal tumour or polycystic kidney.

The fluid of both the above varieties should be removed by open operation if the medical measures alone fail to improve the patient's condition. Most of the patients improve after removal of the fluid as the new exudate that replaces the old is rich in antitoxic and antibacterial material.

The fibro-caseous type of chronic tuberculous peritonitis is quite common. It gives rise to some enlarge-

ment of the abdomen. One can usually feel some indistinct masses in the abdomen and occasionally the rolled-up thickened omentum also. It is these indistinct masses that give the characteristic doughy feel to the hand. These masses are nothing but tuberculous nodules situated within the adherent loops of intestine undergoing caseation and liquefaction. The liquified material may come to the surface and through the abdominal wall usually at the umbilicus or some hernial orifice and give rise to a fæcal fistula. Such a fistula may also occur between two adherent loops of intestine. Such fistulæ, as they are usually connected with small intestine, cause rapid dehydration and wasting and lead to death.

The fibrous type of chronic tuberculous peritonitis results when the patient's resistance to the infection is good. This type produces very little symptoms but may cause sub-acute or acute intestinal obstruction because of intestinal kinking.

The treatment of both the fibro-caseous and fibrous type is purely medical. Operative treatment may be required only for the relief of intestinal obstruction. Mortality after such operations is usually high. The medical treatment is carried out on the same lines as detailed before for other tuberculous conditions.

*TREATMENT SERIES*

## MANAGEMENT OF LEUCORRHŒA

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By leucorrhœa is commonly meant abnormal vaginal discharges other than blood. It may occur at any age, and constitutes the commonest gynæcological symptom, being present in about one-third of all patients consulting a gynæcologist. However, women differ greatly in their tolerance for vaginal discharges. An amount which may be annoying, and seems profuse to one patient, may appear to be acceptable and "normal" to another. The normal vaginal discharge is an admixture of the cervical secretion, transudation from the subepithelial capillaries and lymph vessels of the vagina, and some desquamated cells from the vaginal epithelium. The last gives it a slightly whitish appearance. The normal vaginal fluid is just sufficient in amount to keep the vaginal walls moist and slightly acid in rection (pH 4 to 5) between puberty and menopause.

Any deviation from the normal character of the vaginal discharge may be due to either organic or functional disorders. Abnormal vaginal discharge may vary greatly in its consistency, colour, odour and chemical or bacteriological composition. It may be only an excess of the normal type of vaginal discharge as during pregnancy, or in the different phases of the menstrual cycle. It may be grossly abnormal in both quality and amount. According to the prevailing types of bacteria or other organisms, and the amount of pus, blood or urine in the mixture, it may be white, yellow, grey or greenish. It may be odourless or may have a slight or much offensive odour.

Any abnormal vaginal discharge, requires careful investigation. This is of extreme importance, for leucorrhœa is only a symptom and does not constitute a disease by itself.

A proper investigation of a case of leucorrhœa should start with a careful taking of the history, followed, in order, by external examination, internal examination, and special examinations as and when necessary.

The history is often suggestive. The manner of onset, and the duration of the symptom should be inquired into. Leucorrhœa commencing after a childbirth usually indicates a cervical laceration, or erosion. Subinvolution of the uterus with or without retroversion may also be a cause. Leucorrhœa commencing soon after marriage may suggest gonorrhœa. A similar history may be obtained when the abnormal discharge is due to sexual maladjustment and improper use of contraceptives.

The colour of the discharge should be noted. Is it blood-stained, purulent or frothy? Irritation of the vulva and external genitals is common with trichomonal or monilial infection. In stout subjects with intertrigo, where the vulval area is sodden with perspiration, itching may be present. Pruritus may sometimes be due to a highly acid urine, and more often to diabetes, rather than to the vaginal discharge as such. Inquiry should also be made if the discharge is continuous or varies with the menstrual cycle. The menstrual history should be carefully obtained with regard to the age of menarche, periodicity of the cycle, duration and amount of the flow, presence or absence of pain, and the date of the last menstrual period. History of urinary symptoms is important. Frequency of micturition and dysuria suggest cystitis and urethritis, which, among other things, may be of gonococcal origin.

The previous obstetric history of the patient and history of past illnesses should also be recorded. These may give a clue to some obstetric mishap, or debilitating disease, which have the effect of lowering the general or local pelvic resistance of the patient, and ultimately cause leucorrhœa. The patient should then be subjected to a thorough general examination in order to detect any systemic disease which may be responsible for the abnormal discharge. This should include a hæmatological examination and serological tests if possible.

Before undertaking a gynæcological examination, it is essential to determine whether the patient has been using vaginal douches and/or suppositories. In either case, examination may fail to reveal the true nature and the cause of the leucorrhœa. For at least 24 hours before the examination, no douche or vaginal

medication should be employed. No urine should be voided within 3 hours before the examination is conducted.

Gynæcological examination should be careful and systematic. The external genitals should be inspected first in order to find out the nature and effect of the discharge. The colour, amount and the presence or otherwise of small sticky mucus, froth or lumpy shreds of tissue deserve attention. Excessive vaginal discharge is often the first symptom of cancer and some of the other less serious pelvic non-infective diseases. A watery discharge may result, in part at times, from urethral incontenence, or vesicovaginal fistula. A thin serous or sero-saguinons discharge may be caused by a benign or a malignant growth of the uterus. More rarely, a large hydrosalpinx may intermittently discharge considerable amount of its contents. An abundant greenish yellow purulent discharge is frequently seen in gonorrhœa, although other conditions, for example a badly lacerated and chronically infected cervix with erosion, may sometimes cause a similar type of discharge. A frankly purulent discharge may arise from infection and suppuration at any place along the entire genital tract. A large amount of purulent secretion escaping at one time, or periodically, in a gush, may be due to a pelvic abscess bursting into the vagina. However, more commonly such a condition is created by a gradual accumulation of a purulent secretion in the vaginal fornices, which suddenly escapes owing to changes of posture or sudden contraction of the abdominal or pelvic floor muscles.

A foul smelling discharge in a young woman is usually due to infection associated with incomplete abortion, or placental remains, sloughing fibroid, or a foreign body like a pessary, gauze, cotton or contraceptive tampons. In older women, such offensive discharge may be caused by malignant disease, sloughing fibroids foreign bodies and sometimes senile vaginitis.

A portion of the discharge as it appears at the vulva, should be collected for the examination of its reaction, and also for microscopic study as a stained smear and as a fresh wet cover-slip and hanging drop preparation.

In cases where abnormal vaginal discharge is the main complaint, a speculum examination of the internal organs is an absolute necessity, and should be undertaken before the usual bimanual examination. A bivalve speculum, preferably of the Cusco type is very suitable for the purpose. It is introduced without the use of any lubricant,and a good exposure of the cervix and the vaginal walls obtained under sufficient light. Any excess of discharge is allowed to flow out while a careful note is made of its quantity and characters. Some of the discharge is collected, in a pipette or on a tampon, from the posterior vaginal fornix and should be examined immediately if motile organisms have to be detected. The conditions of the vaginal walls, the fornices and also of the cervix are duly noted. A smear is taken from the cervical canal if necessary. The cervical secretion is obtained by gently squeezing the cervical canal between two blades of a sponge holding forceps. A smear of the urethral discharge after "milking" of the urethra with the finger, should also be taken if venereal infection is suspected.

This examination is followed by a routine bimanual pelvic examination. Special examinations are undertaken for urine, blood pressure, vaginal discharge for trichomonas, amœba or monilia, smears from the cervix or the urethra and perhaps a complement fixation test for gonorrhœa, according to requirements of the case. In presence of urethritis of cystitis a catheter specimen of urine may have to be cultured and the infecting organism determined.

Careful investigation in the manner described above allows the cause of leucorrhœa to fall into one of the four main groups according to its location, in the vulvovaginal region, in the cervix, in the upper genital tract, and finally extragenital areas with constitutional or circulatory disturbances as the underlying cause. The treatment of the leucorrhœa in a patient naturally depends upon the recognition of the cause. In the last group, it is frequently observed that the cause of leucorrhœa is perhaps anæmia, constipation, some debilitating or organic disease or, an imbalance of the endocrine system, particularly affecting the ovaries. In such cases the treatment is to be directed to the relief of the constitutional disorder by appropriate measures, and it is very often found that the leucorrhœa improves with the improvement of the general health. Young girls during puberty and adolescence, complaining of leucorrhœa, often belong to this group. Intensive treatment of anæmia (with proper diet, rest, exercise and judicious doses of iron) and the relief of constipation; is followed promptly by an improvement of both the general and the local condition. Ovarian hormones must never be employed in such cases without a thorough investigation as to the need of these substances. They are potent, but may be harmful when indiscriminately used during the formative years. Instead of sex hormones it may be advisable to give thyroid extract (gr. ½ twice a day) if these girls happen to be of a fat flabby hypothyroid type. Not only young adolescent girls, but many grown up adults are quite often found to suffer from leucorrhœa due to a chronic debilitating disease, profound anæmia, severe constipation, a defective liver or a diseased heart. These patients very often complain that the leucorrhœa is ruining their health. It is often very difficult to convince such patients that the leucorrhœa is not the cause, but the effect of their ill health. A rational treatment for the improvement of the general health cures the leucorrhœa.

As mentioned above, leucorrhœa may be caused by affections of the genital organs other than the cervix, vagina and vulva. Fibroids of the uterus particularly of the submucous variety, specially when it becomes polypoidal often gives rise to an abnormal vaginal discharge. Hypertrophied and polypoid endometrium, may also cause leucorrhœa, although irregular bleeding is a more frequent symptom in both these conditions. Retroversion of the uterus may, occasionally though not as frequently as it is sometimes believed,

cause leucorrhœa. Subinvolution of the uterus and ovarian tumours may give rise to leucorrhœa when these are associated with chronic pelvic congestion. Pelvic inflammatory lesions, specially the chronic and subacute forms (tubo-ovarian inflammatory masses) often cause persistent leucorrhœa. These pelvic lesions can be discovered only after a thorough bimanual examination. The treatment in such cases is planned to remedy the underlying pathological condition. However, a course of six injections of (5 to 10 c.c.) milk (or some other foreign protein) may often be indicated and at least gives some temporary relief from backache which is found as an associated symptom. It must be emphasised that, the mere discovery of a pelvic lesion does not establish the cause of leucorrhœa. A careful search must be made to find out other more probable causes, before an operative treatment is undertaken with the object of curing leucorrhœa for such conditions. This is important particularly where retroversion of the uterus is the only pelvic pathological condition found.

The cervix uteri alone accounts for the largest number of cases of leucorrhœa. Erosion and laceration of the cervix with ectropion are the commonest lesions found, but a cervical polyp also is often responsible. Even in the absence of (existing or previous) inflammation, especially in virgins, the columnar epithelium of the cervical canal sometimes descends and spreads over the area immediately surrounding the external os. This condition, seen in adolescent girls is often termed "congenital erosion." A better name for the condition is "functional erosion" as this is believed to be caused by excessive œstrogenic stimulation of the columnar epithelium of the cervix—a view strongly held by Bourne and others (1937). Diagnosis of erosion of the cervix should never be done by feel alone, but must always be confirmed by inspection with a speculum. Functional erosion of the cervix has often been treated with luteal hormone, but the results are usually unsatisfactory. Improvement of general health and attention to personal hygiene often bring about some improvement, but if persistent, this has to be treated like other varieties of erosion—by electrothermal treatment, either cauterisation, or coagulation. The latter is more satisfactory. Erosion in a parous woman, particularly where signs of previous infection are still present, should receive a course of treatment with penicillin (400,000 units daily) or sulphonamides (1 gm. 4 times a day) for 7 to 10 days before the electrothermal treatment is adopted. Laceration and ectropion, and extensive erosion in a bulky hypertrophied cervix should be treated by suitable operation. In the immediate pre-operative period the patient should receive a course of foreign protein (*e.g.*, milk) injections as indicated above.

The cervical lesion which deserves special attention is carcinoma of the cervix. Although it is not a common cause of leucorrhœa, it nevertheless, is an extremely serious condition. It usually causes only a thin watery discharge which is not often a cause for complaint. It is imperative that any treatment of leucorrhœa of cervical origin must first exclude the possibility of cancer in the cervix, either on the surface or in the depth of the tissues or in the cervical canal. This must be done by a proper elucidation of the history, a thorough clinical examination and, in cases of doubt, by diagnostic biopsy or curettage. If cancer is discovered, the patient must be referred to a competent surgeon or radiotherapist.

The fourth or the last group of cases of leucorrhœa comprises of those patients where the cause of the excessive discharge lies in the vulvovaginal region. Where retained and neglected foreign bodies are the cause, the treatment consists of removal of such foreign bodies. Chemical irritants, used for personal hygiene or for contraceptive purposes, or for douching which is continued long after this has ceased to be necessary or beneficial, may often cause uncomfortable leucorrhœa. The treatment is to discontinue the use of such irritant chemicals along with a mild alkaline (sodium bicarbonate, one drachm to a pint of lukewarm water) vaginal douche under a very low pressure for 4 to 6 days in order to soothe the irritated vaginal mucous membrane. Tuberculous or syphilitic ulcers of the cervix, or of the vulvovaginal region may give rise to leucorrhœa. The treatment is to keep the parts clean with normal saline or lactic acid (one teaspoonful in a pint of warm water) vaginal douches and administration of adequate systemic medicines in suitable dosage, *e.g.*, penicillin in syphilitic, and streptomycin and para-amino-salicylic acid in tuberculous lesions. If a urinary or a recto-vaginal fistula is found to be the cause of leucorrhœa, the treatment is surgical repair of the fistula after preliminary cleansing of the local parts in order to eradicate obvious extraneous infection.

Pyococcal or gonococcal infection is often responsible for a thin or a thick, frequently mucopurulent, leucorrhœic discharge. Vulvovaginitis in children is often, but not always, caused by gonococcal infection. More often the cause is unclean habits, inviting pyogenic infection and sometimes due to the irritation from threadworms and roundworms. Gonococcal infection, if confirmed, requires treatment with penicillin (50,000 units every 3 to 4 hours, continued for one to two weeks). Cleanliness is to be ensured by hot sitz baths with a weak solution of a mild antiseptic (*e.g.*, 1 in 1000 potassium permanganate). The resistance of the vaginal mucous membrane against infection is increased by small doses of œstrogens (*e.g.*, stilbœstrol, 1 mgm. once or twice a day, for one to two weeks). Vaginal douching in children should be avoided as far as possible. In post-menopausal women a whitish, purulent or slightly sanguinous discharge is often caused by senile vaginitis which is due to a secondary infection of the senile atrophic vaginal mucosa. Speculum examination shows small shallow pinpoint ulcers scattered all over the vagina, and a thin pale atrophic epithelium. Treatment consists of small doses of œstrogens (stilbœstrol 0·5 mgm. to 1 mgm. daily) by mouth, local cleansing douches with lactic acid (one teaspoonful in a pint of hot water) along with local application of œstrogen (*e.g.*, Menformon ointment) in an emollient

basis. Bacterial infection causing vulvovaginitis and leucorrhœal discharge is rather uncommon in adults. The use of a specific antibiotic or chemotherapeutic agent is indicated in these cases. In addition, local cleansing is done with low-pressure vaginal douche with normal saline or one teaspoonful of Dettol to a pint of hot water. When gonococcal infection is the cause, often cervicitis is also present. Local cleansing douche under low pressure (never during the acute stage) along with a suitable combination of penicillin (100,000 units, 3 times a day) and sulphadiazine (2 tablets, every 4 hours) usually brings symptomatic relief in a few (5 to 7) days. But a "cure" of the infection is difficult to achieve. King (1947) is of the opinion that the criterion of "cure" is 3 negative smears and an equal number of cultures done at intervals of 2 months, after stopping all treatment.

Protozoal infection of the vulvovaginal region is a fairly common cause of leucorrhœa. Trichomonas vaginalis is often responsible for an infective leucorrhœa although this organism has, on many occasions been isolated from the vaginal discharge in a non-pathogenic condition. Trichomonal vaginitis causes a constant free vaginal discharge which is frothy in appearance and has irritating character. It gives rise to pruritus vulvæ and excoriation in the adjacent parts of the thighs. Diagnosis has to be made by detecting the flagellated organism (first described by Donne in 1836) in a fresh drop of the vaginal discharge. The treatment of trichomonal vaginitis is based on two main principles: (*i*) to bring about a highly acid medium in the vagina which is inimical to the growth of this organism, and (*ii*) local application of some specific chemical to kill the existing organisms as well as to inhibit their further growth. These are attained by giving daily vaginal douches with lactic acid (half to one teaspoonful in a pint of hot water) and frequent introduction of suitable carbohydrate material (glucose, glycogen) in the vagina as available in certain proprietary pharmaceutical preparations (*e.g.*, Devegan tablets). Certain silver (picragol) and some arsenical compounds like stovarsol (*e.g.*, S.V.C. tablets) are employed to kill the parasite. The vagina must be properly cleaned, preferably with ether-soap, and then mopped dry. Vaginal medication is undertaken then, by insufflating silver-picrate powder or inserting picragol suppositories or by nightly introduction of 2 tablets of a suitable proprietory preparation (S.V.C., Devegan or Viotorm tablets). It has been pointed out recently, that the use of acetarsone for the treatment of trichomonal vaginitis, may cause an existing syphilitic infection to pass into a latent stage. Of late, good results have been claimed by McKay and his associates (1949) with the use of aureomycin in trichomonal vaginitis, both by insufflation and as suppositories in a jelly basis.

Prolonged treatment is frequently necessary; for, the symptoms are usually exaggerated after a menstrual period, and re-infection is common from foci in the urethral glands both of the patient and of her husband.

Several cases of amœbic infection of the vagina have recently been reported, specially in this country where chronic amœbiasis is endemic. Presence of amœba alone, however, does not prove that it is pathogenic in the particular patient or that her leucorrhœa is being caused by the amœbæ in her vagina. Treatment consists of injections of emetine, supplemented by cleansing douches and local application of anti-amœbic drugs like enterovioform or carborsone.

Vulvovaginal moniliasis is sometimes found to be responsible for intractable leucorrhœa. The diagnosis has to be made by microscopical examination of the discharge which shows the mycelial threads with the gonidia and the hyphæ. Treatment is fairly simple but has to be carried out with care and thoroughness. One per cent aqueous solution of gentian violet, either alone or mixed with 1 per cent aqueous solution of acriflavine, used for thoroughly painting all areas of the vaginal wall quickly relieves the condition in a few days. Good results have also been reported by the nightly use of 5 c.c. of 3 per cent Ricinoleic acid in a buffered acid jelly or a similar preparation of propionic acid jelly with 0·1 per cent oxyquinoline sulphate (Hesseltine and Beckett; 1949). A vaginal douche with sodii bicarb (1 teaspoonful in one pint of water) is of great help in the treatment of moniliasis.

Roundworms and threadworms sometimes enter the vagina, after their exit from the anus, and bring about an irritative discharge from the vagina, particularly in young girls with unclean habits. The usual treatment for the worms, together with a simple cleansing douche usually clears up the condition in a few days.

From the preceding paragraphs it will be appreciated that leucorrhœa can be caused by numerous conditions, both functional or organic and that the objectives signs and subjective symptoms may vary widely in different individuals even when the ætiology is the same. The treatment also varies widely, mainly directed sometimes to the general health of the patient and at other times to the local lesion and is almost specific in nature.

### References

Bourne, A. W., Bond, L. T. and McGarrity, K. A.—*Brit. M. J.*, **1**:116, 1937.

Hesseltine, H. C. and Beckett, E.—*Am. J. Obst. & Gynec.*, **58**:553, 1949.

King, A. G.—*Am. J. Obst. & Gynec.*, **53**:829, 1947.

McKay, L. V., Laird, R. L., Flanagan, J. B. and Sprunt, D. H.—*Proc. Soc. Exper. Biol. & Med. N.Y.*, **72**:674, 1949.

I

## THE HEALING POWER OF NATURE*

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*Delhi*

At a conference like this there is usually a plethora of technical papers, so it struck me that a short paper on some general non-technical subject may elicit some appreciation. This is my excuse for the venture I have made. But if you will enter deeply into the subject of my choice, *viz.*, "The Healing Power of Nature or *vis medicatrix naturæ* you will, I am confident, agree with me that it is of no lesser importance and is very helpful in practising the art of medicine scientifically. You will also, I am sure, bear me out that a reference to the subject drawing the attention of the profession to it is quite timely in these days for two reasons, *viz.*, (*i*) a distinguished person, with little knowledge of medical science has lately been preaching from the position of eminence he occupies that medicines and injections are not only no remedies but are harmful to health, that nature cure is the safest way to fight disease, that the guiding principle of nature cure is that disease is not disease so much so that fever is itself a medicine and that mankind should shun milk, salt, sugar and several other essential articles of food along with medicines, vaccination, inoculations and injections as a part of nature cure, and (*ii*) the habit of prescribing new, proprietary, rare or scarcely available, often potent, at times almost inert and as a rule highly expensive drugs with little scientific justification, simply because they are the latest in the market, and therefore most fashionable has reached or even excelled the monster-headed poly-pharmacy of pre-Hahnemann days of which many prescriptions abound even now.

In one instance, all told 14 preparations are prescribed. This patient was suffering from poverty of blood and low blood pressure with recurrent profuse diarrhœa and marked wasting.

In my student days I learnt the difference between 'cured' and 'recovered'—two of the modes of termination of illness of a patient. Since then I have kept that distinction in my mind. In the case of those diseases for which we possess specifics—and luckily their number is now fairly large—we are justified in claiming credit for their successful termination while in the case of the rest—a much larger group—the credit must still go to Nature and we may not claim to have done more than having assisted her. A student was once asked to define therapeutics. He answered that therapeutics was a science whereby a doctor claimed credit for what was done by Nature. There is a lot of truth in that though it may not be the whole truth.

Voltaire said that doctors poured medicines of which they knew little into bodies of which they knew less. Since his days the medical science has made great strides, a good deal more is now known about the human body and the drugs, and his dictum has lost much force, but there is no gainsaying the fact that regimen in diet, rest, and exercise, etc. is in many instances better than drugs; and that there is no better general tonic than good food; that hunger is the best sauce, that there is no better appetiser than appetising food and cheerful environments; that exercise is the best digestive; that there is no better restorative of energy than good sound sleep; that nothing is so bracing as sunlight and open air; that there is no better remedy for incurable ills than patience; and that nothing can change a bad constitution.

These facts are always worth remembering. They represent the most important measures by attending to which we can effectively assist and augment the healing power of Nature. And this is really all that can be done in the treatment of those diseases for which there is no specific remedy or radical cure. In such ills Nature, or the body defences as we call them, fights out the battle. The body defences are partly natural, the result of inborn constitution, and partly acquired by the various means of developing acquired immunity and by leading a healthy regular life free from worries and anxieties to the possible extent.

In Nature everything is constantly endeavouring to retain its state and position of stability or normality, and to revert to it if it has been departed from. Take the instance of a salt. It either resists going into solution, or having gone into solution it crystallises out or forms another insoluble compound the moment the circumstances become favourable for it. As another instance take a person convalescing from enteric fever. He often develops voracious appetite and puts on flesh sometimes in excess of his pre-disease level. He does not need an appetiser or digestive or tonic, and if you do give him some such thing it would be just to satisfy his and his attendants' desire and may be also your own, for the magic—the mysticism—which is inherent in a bottle of medicine.

A shift from normal to abnormal in Nature is more difficult than *vice versa*. That is why innumerable noxious agents notwithstanding we so seldom fall ill and when we do we so often and so easily get well. Perhaps the best example of the healing power of Nature working side by side with the noxious agent, and in many instances overcoming it in the end, is afforded by tuberculosis. It is Nature functioning through the body defences that cures this disease. All therapeutic measures, medical, surgical, dietetic, climatic and so on and so forth on which we have relied so far, are nothing but aids to her. Nature is laying down fibrous tissue at the periphery of a focus as the destructive process is going on in its centre. It is this fibrous tissue which ultimately seals the focus and walls in the microbes. For this purpose it is essential for the body defences to be maintained at a high pitch by judicious living and to be strengthened and fortified with the aid of the various measures already mentioned. The significance of calcification

*Read at the Scientific Section of the 27th All-India Medical Conference, Sholapur, Dec. 1950.

in this disease as a means of healing has been overrated. It is not a sign of healing but a by-product of caseation. Once the disease is well established the incrustation of caseous foci with calcium salts has probably little effect on the final outcome. In fact, calcification as it occurs in tuberculosis does not differ from calcification in any other tissue. The calcium salts are deposited in tuberculous foci in accordance with the universal principle that necrotic tissues, or for that matter any non-living permeable material, whether dead tissue or avascular hyaline connective tissue or extraneous foreign material such as silk sutures and gauze drains etc. which cannot be absorbed will get impregnated with calcium salts. Why this should be so is not yet understood but it explains the futility of saturating a tuberculous patient with calcium salts, whether by mouth or parenterally.

The best instances I can quote of Nature *cum* Faith Cure is that of the world famous clinic of Rollier at Leysin in Switzerland which I visited many years ago—a clinic of about 1,200 beds for surgical tuberculosis, all in private hotels catering for patients of all economic grades, all very neat, clean and hygienic, all giving ample, wholesome and well-balanced food, the most expensive ones providing every conceivable luxury and the least costly shorn of it, all offering facilities for recreation, relaxation and amusements, all looking to the entire comforts of their clientele, all fitted for heliotherapy, all patients taking the sun cure with religious zeal, regularity, punctuality and precision with full faith in the miraculous healing power of the sun, all starting treatment with the belief that 2 years was the minimum period required for "Cure", all visited by Rollier about once in 4 weeks, some for a few seconds, others for a few minutes with an invariable smile and a word of cheer and comfort, his patent saying being "Patience is the best remedy", and none taking a grain of calcium or a drop of cod-liver oil. Vitamins had not come in then, but even if they had, I am sure, they would not have formed a part of the healing balm of Rollier. The clinic presented a wonderful sight and produced results unequalled elsewhere in the world. I happened to ask Rollier to recommend an ultra-violet lamp for use in India. He at once silenced me by saying "When you can get the natural why do you run after the artificial".

What the natural body defences are, how they are built and what they are dependent on it is not my purpose to relate, nor would I place the reticulo-endothelial system or the endocrine glands or any other system over and above the other organs in this respect. A healthy and harmonious working of all the tissues collectively is essential for the body economy. It has been well said that the God Brahma has hidden the mystery of the man in his own body although astrologers, alchemists and philosophers have sought for it outside the body.

It is not my intention to develop the technique and details of the various therapeutic measures we adopt to assist Nature. I have named the most important ones and I will leave the subject at that.

Some of you might say: "But our patients are not satisfied unless we give them a bottle of medicine", and it is so, for from time immemorial physician, patient and bottle have formed an inseparable trio. In my practice I have often been called a therapeutic nihilist, but I have never believed or preached that drugs are of no use and I most strongly dissent from Shakespeare when he makes Timon of Athens say to the thieves:

"Shun the physician
For his antidotes are poison, and he slays more
than you rob."

What I have been and am against is the indiscriminate and unscientific use of drugs and hold that no therapeutic measure, no matter how innocent, is to be used unless there is a scientific indication for it. I do not preach "masterly inactivity" and I know "masterly inactivity" is no easy doctrine when it comes to be applied in practice.

In the case of specific drugs the necessity for their use is, of course, obvious—nay imperative. It is the other drugs I have been talking of. Broadly speaking, they have two uses: one to relieve certain distressing symptoms and the other to produce suggestive effects on the minds of the patients, and may be also on those of their attendants. For the first purpose you will no doubt choose appropriate drugs; for the second purpose, *viz.*, as a vehicle of suggestion, I would implore you to prescribe inert harmless and inexpensive substances and change their colour, taste and flavour as often as you like. The not too uncommon practice of prescribing very costly proprietary medicines, in preference to equally effective pharmacopœial preparations, without any scientific reason is highly detrimental to the small purse of our patients, the limited resources of our poor country and the reputation of the scientific system of medicine. A tremendous amount of money which is being wasted on un-essential and un-indicated drugs could be saved for use on good food, suitable residence, proper nursing and many other equally useful amenities of life so essential for a patient.

The bottle, I admit, will never be abolished, but let us not make it the be all and end all of treatment, and in prescribing its contents let us bear in mind their limitations. My experience has convinced me that a simple explanation of the nature of the patient's illness, and of the extent to which drugs and other measures can be useful in its treatment, obviates in many cases the necessity of prescribing unnecessary and useless medicines, and certainly goes in all cases a longer way to satisfy the patient than the bottle of medicine.

There are illnesses which drugs cannot cure but Nature can. Drugs, however, can, if judiciously used, aid Nature and give relief to the patient. Our duty—rather moral obligation—is to instruct the public in matters of health and to tell what drugs are good, what are useless—may be even harmful—and what can be achieved by wisely assisting Nature in her wonderful performance. Sydenham said: "It is a great mistake to suppose that Nature always requires the

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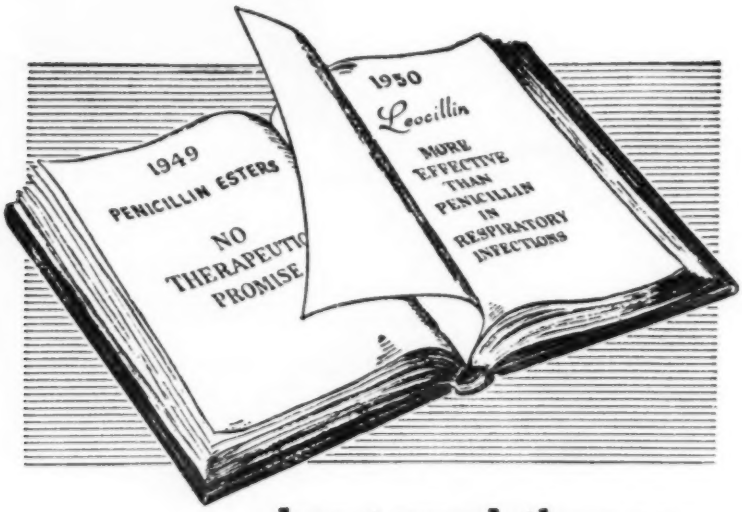


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assistance of art." This points to the great moral, often overlooked, that it is ultimately Nature whose aid must be relied upon to cure, whatever methods of assisting her may be employed. If we go back to Hippocrates we find that he laid a sound foundation for therapeutics by teaching that "our natures are the physicians of our diseases". But his professed regard for the *vis medicatrix naturæ* was not shared by his immediate followers, who formed themselves into various sects, such as Dogmatists, Methodists or Pneumatists. Among the methodists, for example, was Asclepiades. He completely denied the healing power of Nature and asserted that the physicians must grapple actively with the disease and cure it "safely, swiftly and smoothly". A physician following in the footsteps of Asclepiades, however dangerous in reality, is often highly popular and fashionable. But he is not the modern scientific physician and is not to be emulated. Hippocrates' teaching ought to be the ideal to follow, and we should continue to learn from and depend upon Nature when we have no sure weapon in our scientific armamentarium.

## II

## BLOOD COMPATIBILITY TESTS

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In the last ten years considerable advances in knowledge in blood group serology have emphasised the potential hazards of blood transfusion. These advances have made it essential that blood compatibility tests should be conducted in such a way as to ensure that every possible step is taken to prevent false results.

Every transfusion requires prior proof of the compatibility of the donor's blood with that of the recipient, even if the blood of the same donor has been shown to have been compatible with that of the recipient at a previous transfusion.

It should be appreciated that compatibility of transfused blood is essential not only to avoid the ill-effects of incompatibility, but also to avoid sensitising the recipient, so as to render subsequent transfusions dangerous. This can occur in respect of antigens such as those of the Rhesus system, the antibodies of which are not normally present, but may be produced by transfusion of red cells containing the antigen to a recipient who lacks it. This is particularly important in the case of females of any age up to the time of menopause, because if they are sensitised they may not be able to bear live children. The ideal, therefore, is to always transfuse blood of identical erythrocyte antigen composition. Few laboratories, however, have a sufficient range of antisera for this purpose. Under the circumstances the following procedures are considered best for general use.

Compatibility tests are either laboratory (*in vitro*) tests or biological (*in vivo*) tests. The laboratory compatibility test is commonly called a cross-match test. This test determines the interaction between the donor's cells and recipient's serum as well as that between the donor's serum and recipient's cells. The former is more important because if the donor's cells are agglutinated by the recipient's serum after transfusion there may be several unpleasant sequelæ. Agglutination of the recipient's cells by the donor's serum *in vitro*, on the other hand, is not so important because, in the great majority of cases, agglutination will not take place *in vivo*, partly because of the dilution of the donor's serum in the recipient's blood, and partly because cross-absorption of agglutinins in the donor's serum takes place between the antibodies in the donor's serum and group specific substances in the recipient's serum, so that they have no effect on the recipient's red cells. It should be borne in mind that rarely the donor's serum may contain high titre antibodies *e.g.*, the "dangerous universal donor" in which case a fatal issue may result from the action of the donor's serum on the recipient's cells. Blood which, therefore, contains antibodies active against the recipient's red cells should be administered only in an emergency *i.e.*, for cases of acute hæmorrhage, but not for the transfusion of non-urgent cases, such as anæmias.

The possible results of a cross-match test and their interpretation is summarised below:

| Donors' cells | Recipients' cells | |
|---|---|---|
| + | + | |
| Recipients' serum | Donors' serum | |
| − | − | Blood perfectly compatible |
| − | + | Can be used in an emergency only. |
| + | − | Definitely incompatible. |
| + | + | Definitely incompatible. |

+ = Agglutination.
− = No Agglutination.

In performing the cross-match test for a non-urgent transfusion to a person who has been transfused before, or, for a first transfusion to a pregnant woman, several technical precautions need to be taken. These are outlined below:—

(1) Tests must be performed in tubes as interactions associated, for example, with the Rhesus groups are ordinarily not demonstrable on slides and tiles.

(2) A 1-2 per cent suspension of red cells should be used. The use of stronger suspensions may result in agglutination not being detected.

(3) Red cells after two or three washings in saline should be suspended in a protein medium. The best protein is 20 per cent bovine albumin (Fraction V) prepared by Armour Laboratories. If this is not available serum of group AB can be used as a suspension medium. The use of a protein suspension medium will ensure that the albumin type of Rhesus antibody will not be missed. This type of antibody does not act on cells suspended in a saline medium unlike the saline type of Rhesus antibody which can act upon cells suspended either in saline or albumin.

(4) Serial dilutions of serum should be used as zoning may be encountered *i.e.*, undiluted sera may not act, whereas diluted samples of the same serum will bring about agglutination.

(5) Cell-serum mixtures should be kept in an incubator at body temperature for two hours.

(6) All tests should include a control consisting of the patient's cells against his own serum. This serves to exclude false positives due to auto-agglutination.

(7) All results should be read under the low powers of the microscope after carefully pipetting off a little of the cell deposit on to a slide. Naked eye examination of the cell deposit has been found to be misleading. Further, rouleaux formation can be distinguished from true agglutination.

(8) Cells which are not agglutinated should be tested for adsorbed antibody by the method of Coombs, *et. al.* (1945). This test is done by making a 1 per cent saline suspension of the cells. These are washed five or six times, and resuspended in saline so as to make a 5 per cent suspension. A drop of this is mixed with a suitably diluted rabbit anti-human globulin serum, allowed to stand for a minute or two, and then rocked very slowly. When positive, macroscopic agglutination appears within ten minutes. Two controls, one with saline instead of antiserum, and the other with normal cells instead of the test cells should be put up.

A positive result indicates that antibody globulin has been adsorbed on to the surface of the cells, which are, therefore, incompatible with the recipients' serum.

In spite of these numerous measures to exclude all possible sources of error, occasional incompatibility reactions result. For this reason biological (*in vivo*) tests for incompatibility have been advocated. The most satisfactory biological test is that advocated by Weiner *et al.* (1942). This test is performed as follows:—

100 c.c. of the prospective donor's blood is mixed with 10 c.c isotonic sodium citrate solution. 10 c.c. of blood are withdrawn from the prospective recipient and divided between two test tubes, one empty, the other containing 1 c.c. of isotonic sodium citrate. Through the same needle 50 c.c. of the donor's blood is injected with a syringe. After an hour another 10 c.c. of blood is withdrawn from the patient and is treated like the first 10 c.c. The citrated samples are centrifuged and the colour of the supernatant plasma in each is compared. The sera separated from the clots in the empty tubes can be used later as control. If the injected cells are incompatible, the patient may shiver and there may be a rise in his temperature. Often, there are no symptoms at all, nevertheless, the second sample will always show an increased icteric index as compared to the first sample, though the icteric index will be within normal limits. If time permits, the remaining 50 c.c. of donor's blood can be injected, and the test repeated as an additional safeguard. This test is unsuitable, for obvious reasons, if intravascular hæmolysis from any cause is taking place, or if the donor's blood is not fresh. The syringes used should, of course, be perfectly dry, or should be sterilised by boiling in isotonic saline and not in water because hæmolysis of red cells will then take place in the syringe.

## Conclusions

Rapid advances in knowledge of blood group serology have emphasised the value of careful compatibility tests prior to transfusion. These are of two types (*a*) laboratory (*in vitro*) tests and (*b*) biological (*in vivo*) tests.

It must, however, be emphasised that the biological test is not to be considered an alternative to a laboratory test done as outlined above, because such a laboratory test will serve to exclude all but the rarest incompatibility reactions. The biological test should be undertaken if no suitable laboratory facilities exist.

It should be evident that the skill and experience required for the satisfactory conduction of the laboratory test can no longer reasonably be expected of the 'clinical side-room.' This constitutes one good reason for the establishment of central blood banks staffed by specially trained personnel.

## References

Coombs, R. R. A., Mourant, A. E. and Race, R. R.—*Brit. J. Exp. Path.*, **26**:255, 1945.

Fulton Roberts, G.—The Rhesus Factor. William Heinemann, London, 1949.

Wiener, A. S., Silverman, I. J. and Aronson, W.—*Am. J. Clin. Path.*, **12**:241, 1942.

Postscript—Since this paper was written, Cochrane *et al.* (*Brit. M. J.*, **2**:1203, 1950) have described an anti-Kell antibody which reacted in saline but not in albumin. It would therefore be advisable to carry out compatibility tests in both media.

# CASE NOTES

## THE AZYGOS LOBE OF THE LUNG

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Only one case of azygos lobe of the lung was found on routine investigation, during three years of service at a combined general hospital. Considering its rarity, it is worth recording the case.

Originally described by Wrisberg (1778) as quoted by Cairney (1923), the azygos lobe was only an anatomical curiosity till 1923. It is formed by the division of the right upper lobe into two unequal parts by the azygos vein which draws a fold of the parietal pleura during the early development of the heart. This so happens particularly if the communicating branch from the right upper limb does not disappear, as it should do normally, during the descent of the heart into the mediastinum. The thin curvilinear shadow in the radiogram is due to the two layers of the pleura at the base of which is situated the azygos vein. The lobe of the azygos vein is usually found on the right side; but its occurrence on the left in relation to the left superior intercostal vein is also recorded as quoted by Raman (1944). Again this should not be confused with the azygos lobe described in books on human embryology and morphology. The latter is a detached part of the right basal lobe occupying the sinus subpericardiacus and is a sign of atavism. Stibbe (1919) classified the azygos vein according to the position of the fissure into three types, called A, B and C. Type A—where the fissure is horizontal and cuts the lateral surface below the apex; Type B—practically vertical and cuts the apex; and Type C—where the fissure is vertical and cuts a tongue-shaped lobe from the medial surface. Mathes and Coope (1928) state that the aberrant azygos vein may cause a dense comma-shaped shadow. Nelson and Simon (1931) described it as a horse-shoe-shaped shadow. Raman (1944) reviewed the literature, discussed the etiology and added two cases to the records. In the review he comments on the rare incidence of cases. Only two cases were recorded in King George Hospital, Vizagapatam, over a period of ten years. None were recorded in the anatomy theatre or the post-mortem records of the same place. Viswanathan (1944) discussed the development, reviewed the literature and reported two cases, one of which was associated with tropical eosinophilia. According to him, up to that time only about two hundred cases were on record. Underwood and Tattersall (1933) suggested heredity as an etiological factor. Raman (1945) in his report of five cases gave an account of detailed radiological examination of relations of two of his patients, which proved negative. Rothstein (1949) describes with radiograms, cases where azygos lobe was involved in disease processes and also the pseudo-azygos lobe which might confuse the diagnosis. He described a case of pulmonary tuberculosis affecting the azygos lobe alone. In the introduction he states that the azygos lobe is too common to need any mention unless it is involved in a pathological process. This is in marked contrast to the experience of the Indian writers (*loc. cit.*). Probably this anomaly is not so frequent in India.

### Case Report

A Hindu, male, aged 23 years, was admitted for cough and intermittent fever of three months' duration. He had no hæmoptysis and there was no marked loss of weight.

*Physical examination*—A well-built adult male, having no cyanosis or clubbing of the fingers; not breathless. Chest showed bronchovesicular breath sounds with basal rales on the left side. Otherwise nothing abnormal was found. Examination of the other systems revealed nothing significant. Routine examination of urine, stools, sputum etc., showed no abnormality. Radiogram of chest showed an accessory lobe of the right lung and increased mottling of lower and midzones on both sides with a suspicious cavity on the left side. There was no evidence of tuberculosis. The accessory lobe was not involved in any disease process. It was of Stibbe B type (*vide* Plate).

### Summary

1. A case of azygos lobe of the right lung recorded.

2. A brief review of the literature made.

### References

Cairney, J—*J. Anatomy*, **58**:54, 1923.
Mathes, J. H. and Coope, R.—*Brit. J. Radiol.*, **1**:481, 1928.
Nelson, H. P. and Simon, G.—*Brit. M. J.*, **2**:9, 1931.
Raman, T. K.—*J. Indian M. A.*, **13**:98, 1944.
Raman, T. K.—*The Indian Physician*, **4**:45, 1945.
Rothstein, E.—*Am. J. Roentgenol. Radiol.*, **61**:195, 1945.
Stibbe, E. P.—*J. Anat. Physiol.*, **53**:305, 1919.
Underwood, E. A. and Tattersall, H.—*Tubercle*, 1, 1933.
Viswanathan, R.—*Indian M. Gaz.*, **53**:518, 1944.

## STRANGULATION—CAUSING INTRAUTERINE FOETAL DEATH IN EARLY PREGNANCY

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B. S., Hindu female, aged about 18 years; gravida ii, para nil was admitted in the Hospital on 10-6-51 at 6 A.M. with amenorrhœa of three and half months duration, slight lower abdominal pain and vaginal

bleeding for 2 days. Last pregnancy had terminated in abortion at the third month, one year ago.

On examination, the height of the fundus was 1" above the symphysis pubis, the os was closed and there was a mucosanguinous discharge per vaginum. The condition was diagnosed as threatened abortion and the patient was put at rest in bed with Progesterone I.M. and bromides and vitamin E per mouth. The patient was well for about 24 hours after which the pain recurred with a greater intensity and a moderate amount of hæmorrhage. The os was one finger dilated. After about 4 hours, on 11-6-51, at about 10 A.M. the products of conception were expelled which on closer examination revealed the following:—

(1) The placenta and membranes were normal. The placenta was 3" in diameter and ½" thick. The placenta weighed 37·5 gm.

(2) The umbilical cord was 27 cm. long and about 3 mm. in diameter and pale white in colour. From the umbilicus it ascended over the left shoulder and making one and a half turns around the neck of the fœtus was inserted centrally in the placenta. The twist round the neck was very tight and was adherent to the neck by a thin and delicate layer of membrane. The neck was very much narrowed and grooved in the region.

(3) The fœtus was 11·5 cm. long and weighed 34 gms. The head was normal with openings for the eyes and mouth, and a rudimentary nose was present. The skin was brownish-white in colour and glistening. The abdomen was big containing a little free fluid and some putrefactive gases. The skin over the abdomen showed some patches of discoloured areas suggesting early putrefactive changes. The limbs showed fingers and toes but no evidence of nails. Rudimentary genital (phallus) was present.

In the absence of any other gross abnormality and the expulsion of a strangulated fœtus with a grooved neck and the cord attached to the neck by a delicate membrane (*vide* Plate), it would appear that the strangulation was antemortem and probably incidental to cause intra-uterine death of the fœtus resulting in abortion.

SUMMARY

1. A report of a case of abortion of a strangulated fœtus is presented.

2. Strangulation by a long cord as a rare but probable cause of intra-uterine death of the fœtus in the early months of pregnancy and consequent abortion is suggested.

ACKNOWLEDGMENT

My thanks are due to Dr. R. Banerjee, B.SC., M.B., F.S.MF., 1st Gynæcologist and Obstetrician, B.S.M.S. Hospital for his valuable suggestions in the compilation of these notes.

## ATRIAL SEPTAL DEFECT WITH MULTIPLE CONGENITAL ANOMALIES

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Great interest has recently been aroused in congenital heart diseases owing to application of newer methods of investigation like angiocardiography and cardiac catheterization and relief of symptoms by surgery as in Fallot's tetralogy, the commonest variety of cyanotic type of congenital heart disease, patent ductus arteriosus, coarctation of the aorta and valvular type of pulmonary stenosis. Though the causes of congenital heart disease remain undetermined, the association of virus infection of the mother, particularly rubella during the early months of pregnancy and congenital heart lesion in the new-born baby has been fairly common to be of etiological significance.

Without minimising the importance of angiocardiography and cardiac catheterization it may be said that most of the congenital heart lesions can be diagnosed from history, physical findings, electrocardiogram and skiagram of the heart and chest (Sellors, *et al.*, 1950).

Of the congenital heart diseases, atrial septal defect is one of the commonest. As a single congenital lesion it constitutes 7-25 per cent of such cases (Bedford *et al.*, 1941). It is often associated with other congenital cardiac lesions like patent ductus arteriosus, ventricular septal defect, pulmonary stenosis, common atrioventricular canal, transposition of arterial trunks, Fallot's tetralogy; in fact, it may accompany almost any congenital lesion (Bedford *et al.*, 1941). Roesler (1934) emphasized the under-development, frail body and weak constitution in patients with atrial septal defect and Abbott (1937) mentioned about Mongolian idiocy and various skeletal anomalies in patients suffering from congenital heart lesions. These included among others arachnodactyly, accessory spleens and pancreas, hydrocephalus, harelip, cleft palate, branchial cleft, dermoid of eye, rudimentary ear, cystic kidney, coloboma of iris, suppression of ribs, imperforate anus and talipes varus. Bedford *et al.* (*loc. cit.*) in their 53 cases of atrial septal defect found underdevelopment in 8 but did not notice Mongolian idiocy or other developmental defects in any of them.

The left atrium being situated at a higher level and the pressure inside it being greater than that in the right atrium, the flow of blood is from left to right atrium. Hence cyanosis does not appear till the flow of blood is reversed due to increased pressure in the right atrium. This may occur in late cases showing failure of the right side of the heart. The disease usually remains latent till failure supervenes, in course of time or is precipitated by pulmonary infection. Dyspnœa then becomes a main symptom. The malady is much more common in the female sex, the ratio between female and male sexes being about 2:1 (Wood,

FIG. 1—Skiagram in P. A. position showing generalised enlargement of heart, prominent pulmonary arc, marked pulmonary congestion and bilateral cervical ribs, rudimentary on right and prominent on the left.

FIG. 2—Skiagram in R. O. A. position showing prominent right ventricle and pulmonary artery. No enlargement of left atrium.

FIG. 3—Skiagram showing marked decalcification of bones of the reported case.

FIG. 4—Skiagram of normal bone of a female of the same age as in Fig. 3 for comparison.



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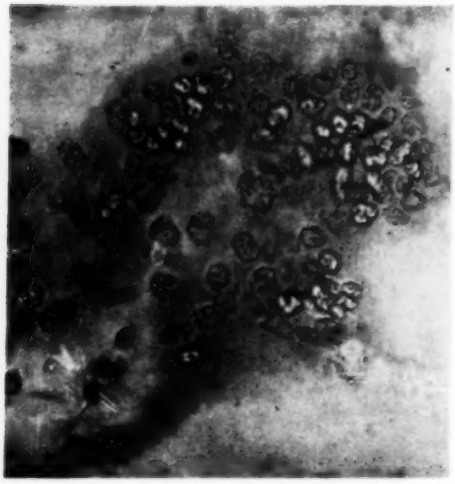
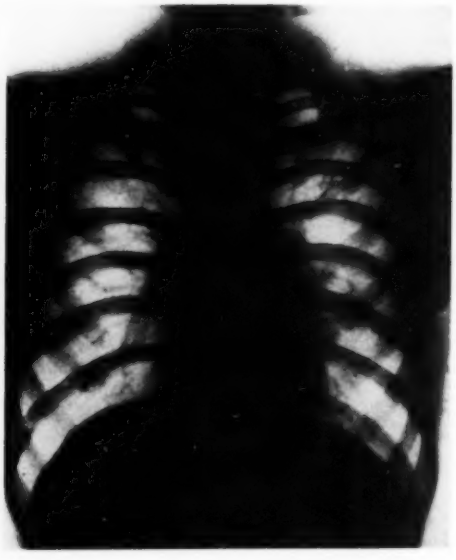


FIG. 5—Tip of chorionic villus from 10 week placenta. Stained Gomori's method for alkaline phosphatase. Note absence of any reaction.

FIG. 6—Chorionic villus from normal full-time placenta. Note black line indicating presence of alkaline phosphatase in syncytium. A second deposit is sometimes present at the base of the cells.

MUKHERJEE—*Plasma Alkaline Phosphatase in Toxaemia of Pregnancy.*



Radiogram showing azygos lobe of the right lung (Stibbe B type).

RAO—*The Azygos Lobe of the Lung.*

Photograph showing strangulated foetus with a grooved neck and the cord attached to the neck by a delicate membrane.

MUKHERJEE—*Strangulation—Causing Intrauterine Foetal Death in Early Pregnancy.*

1950), 3:1 (Bedford *et al.*, 1941). The pulse is usually small and the blood pressure is low. The apical impulse is displaced due to enlargement of the right ventricle. The other characteristic features are due to marked dilatation of the pulmonary artery. These are: a forcible systolic pulsation often with a systolic thrill and a diastolic shock and a dull note on percussion over the second left intercostal space, and a pulmonary systolic murmur and markedly accentuated pulmonary second sound. It should be noted that the defect itself has no characteristic sign of its own and no murmur is usually produced by the passage of blood from left to the right atrium.

A systolic murmur is often heard over the mitral area. A diastolic murmur may be noted over the pulmonary area due to pulmonary incompetence. A frequent combination is mitral stenosis along with atrial septal defect (Abbott, 1915 and Lutembacher, 1916) when the diastolic murmur of mitral stenosis will be heard over the mitral area. On fluoroscopy there are generalised enlargement of the heart due to enlargement of right ventricle and atrium and dilatation of pulmonary artery and its branches. Pulsations are often seen over the right pulmonary artery and hilar dance may be noticed. The skiagram of the heart shows enlarged pulmonary artery, right ventricle and right atrium, a hypoplastic aorta and marked hilar congestions, the right pulmonary artery often showing as a comma. The electrocardiogram shows large and high P waves, sometimes these being bifid, marked right axis deviation, inversion of T waves in leads II and III and widening of QRS complex suggesting bundle branch block.

Occasional cases may live up to old age, but most patients die in the fourth or fifth decade of life. Average age at death in Roesler's (1934) series of 62 cases was 36 and in the cases reported by Bedford *et al.* (1941) the age at death was between 30 and 50. In their series of 53 cases the causes of death in 10 patients were congestive cardiac failure in 3, pulmonary infarction in 2, embolism (one paradoxical) in 2, subacute bacterial endocarditis in 1, bronchopneumonia in 1 and operation in 1. It may, however, be noted that incidence of subacute bacterial endocarditis is very uncommon in this type of congenital heart disease. Auricular fibrillation may set in in late cases and the association of mitral stenosis with this malady has already been stated.

The present case is interesting as in addition to atrial septal defect the patient suffered from infantilism and has a left supernumerary nipple, bilateral cervical ribs and blue sclerotics. There was also generalised decalcification of bones. To the author's knowledge this combination has not been reported before.

## Case Report

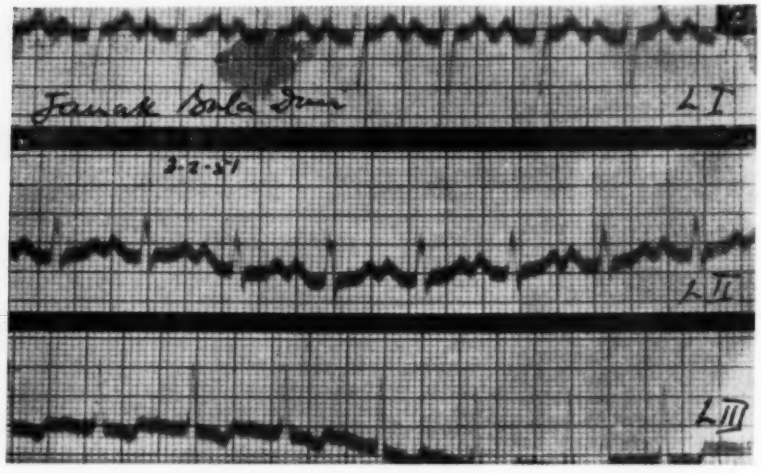
J. B., female, aged 18 years, was admitted under me on 26-12-50 for marked dyspnœa, precordial pain and cough of 15 days' duration. The patient first noticed one year ago that she was getting breathless on exertion. Since then she had suffered from occasional attacks of cough, cold and fever with exacerbation of dyspnœa. Her lips or finger nails never became blue. There was no history of hæmoptysis. The cough was dry. She had not yet menstruated. She was married 5 years. There was no history of rheumatic infection. She was the only child of her parents and the mother died of diseases unconnected with heart when she was five. The patient never sustained any fracture. No history of osteogenesis imperfecta could be obtained in other members of the family. The patient was markedly breathless and had to be propped up. Nutrition and development were very poor. There was no cyanosis, clubbing of the fingers, jaundice or œdema. Slight anæmia was present. Neck veins were not engorged or pulsating. There were no glandular enlargements. Sclera of both eyes were of faint blue colour. Temperature was 102°F., pulse 130, and respiration 40 per minute. The præcordium was bulging and there was a supernumerary nipple on the left side. The apical impulse was felt in the sixth left intercostal space in the anterior axillary line. It was regular in rhythm and slapping in character. There was a diffuse pulsation over the præcordium and a forceful thrust over the lower end of the sternum and the area left to it. Systolic pulsation with a systolic thrill was felt over the pulmonary area. Both right and left borders of the heart were outside normal limits and a zone of dullness was present in the left second intercostal space. A systolic murmur was present all over the præcordium which was loudest at the pulmonary area. There was slight upward propagation of the murmur. The mitral systolic murmur was propagated towards the axilla. Pulmonary second sound was markedly accentuated. No diastolic murmur could be heard anywhere. Blood pressure on admission was systolic 90 mm. and diastolic 52 mm. of Hg. Later, the systolic pressure varied between 86 and 104 and diastolic 70 and 54 mm. of Hg. *Lungs*—scattered rhonchi and râles were heard over both lungs. *Liver* was enlarged two fingers breadth below costal margin. It was soft and slightly tender. *Spleen* was not palpable. There was no free fluid in the peritoneal cavity. Her mental functions were normal and no abnormalities were detected in the nervous system. *Breasts* were not developed and there was no hair over the axillary and pubic regions.

2-1-51. Screen examination:—Heart greatly enlarged, bulging of the pulmonary arc; marked hilar congestion; hilar dance present. Skiagrams: Marked pulmonary congestion; pulmonary conus very prominent; the right ventricle and the right auricle enlarged; no left auricular enlargement; some enlargement of the left ventricle; cervical ribs present on both sides, more prominent on the left (Figs. 1 and 2, *vide* Plate).

8-2-51 The gynæcological specialist reported: Breasts—hypoplastic; tricosis very scanty, hymen—intact; clitoris—normal; P.R. examination—uterus very small, hypoplastic.

10-2-51. Electrocardiogram: Sinus tachycardia. Axis deviation—marked right. P.R. interval—0·16 sec. QRS—duration 0·08 sec. Right bundle branch

block which is frequently found in atrial septal defect was not seen in this case.




ELECTROCARDIOGRAM TAKEN ON 10-2-51 SHOWING SINUS TACHYCARDIA WITH RIGHT AXIS DEVIATION

28-2-51. Hb. 10 gms. per 100 ml. of blood; W.B.C.—8,600 per c.mm.; poly—68 per cent, lympho—26 per cent, mono—2 per cent and eosino—4 per cent. No abnormalities were detected either in the urine or the stool.

2-6-51. Skiagram of both forearms and fingers of both hands—marked decalcification of all bones (Figs. 3 and 4, *vide* Plate).

*Clinical course*—With rest, oxygen inhalation, penicillin 50,000 units intramuscularly every 4 hours and tincture digitalis 20 minims every 6 hours, the dyspnœa was less and the patient became more restful. The temperature settled to normal on the sixth day after admission, the penicillin, however, was continued for another 5 days. It took 12 days for the pulse and respiration to come to normal level.

While in the hospital, the patient on 18-1-51, had another bout of fever with cough and dyspnœa. The temperature went up to 102°F and the pulse rate rose to 130 and respiration to 48 per minute. There were scattered rales and rhonchi on both sides. She was again put on penicillin. This irregular temperature often rising to 100°F persisted from 18-1-51 to 27-1-51. This time she had no digitalis. Though the temperature came down to normal, the respiratory rate was 24 and pulse rate 100 per minute. She was still weak but could walk slowly on the flat without difficulty.

### SUMMARY

1. A case of atrial septal defect with infantilism, supernumerary left nipple, bilateral cervical ribs, blue sclerotics and marked generalised decalcification of bones has been described.

2. Importance of secondary infection particularly of the respiratory tract in the precipitation of cardiac failure has been stressed. The patient was observed during two such attacks and penicillin was very helpful on both the occasions.

### ACKNOWLEDGMENTS

I am grateful to Dr. A. K. Dutta Gupta, M.B., D.T.M., Superintendent, Nilratan Sarkar Medical College Hospitals for the permission to publish this case, to Dr. C. L. Mukherjee, M.B., M.O., M.R.C.O.G., PH.D. for the gynæcological opinion, to Dr. A. K. Bose, M.SC., M.B., M.R.C.P. for the report on the electrocardiogram, to Dr. R. Roy Choudhuri, for the radiological reports and to Drs. A. N. Sen Gupta and S. Mitra for the clinical notes.

### REFERENCES

ABBOTT, M. E.—*Bull. Internat. Assoc. Med. Mus.*, **5**:129, 1915.
*Idem*—Congenital Heart Disease in Nelson's New Loose Leaf Medicine, 18th Edition, 1937. Vol. IV, p. 207.
BEDFORD, D. E., PAPP, C. AND PARKINSON J.—*Brit. Heart J.*, **3**:37, 1941.
LUTEMBACHER, R.—*Arch. d. mal. du coeur.*, **9**:237, 1916.
ROESLER, H.—*Arch. Int. Med.*, **54**:339, 1934.
SELLORS, T. H. *et al.*—*Lancet*, **2**:887, 1950.
WOOD, P.—*Brit. M. J.*, **2**:693, 1950.

## A CASE OF POLYSEROSITIS (TUBERCULAR) TREATED SUCCESSFULLY WITH STREPTOMYCIN

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### CASE REPORT

An Indian Male, 28 years, was admitted in the East Medical Ward of the V. J. Hospital, Amritsar on 16-4-1949 with the following complaints:

Fever for 20 days, pain in the upper half of abdomen for 7 days, breathlessness for 3 days and pain in the back for 6 days.

*Past illness*—Suffered from continuous fever for about 14 days about 7 months ago.

*Personal and family history*—Married; wife healthy who had one premature delivery. Mixed type of diet.

*History of present illness*—About 27 days back the patient began to prepare himself for taking part in a tournament. He used to take part in high and long jump. About 25 days back while negotiating a long jump, his feet struck against hard ground and he felt slight pain in the epigastrium, right and left hypochondria which subsided after a few minutes. After this he did not feel like taking exercise and stopped the practice. About 20 days back he developed fever, the temperature was only 99°F. After this the temperature increased gradually and in a week ranged between 100° and 101°F and he continued like this till admission to the hospital. About 6 days back he developed a dull pain in the epigastrium and both hypochondria. About 3 days back he developed breathlessness and became restless. There was increased secretion of saliva since 3 days. He also developed œdema of the face 3 days ago.

*Physical Examination*—Well built, well nourished youngman, anxious expression, marked dyspnœa. B. P.—90/60 mm. of Hg. Temperature—100°·8F. Pulse—90 per minute.

Circulatory System: Pulse—pulsus paradoxus, pulse improves during expiration and becomes imperceptible during inspiration.

Neck veins are full, when the patient is lying down, become less prominent when he sits up. They fill from below. One gland is enlarged in the supraclavicular fossa on the right side.

Heart: There is pitting œdema over the præcordial region. Apex beat not visible. Percussion reveals that the area of cardiac dullness is much increased on both sides. The right border of the heart is about one finger beyond the right border of the sternum. The left border goes upto the left anterior axillary line in the 5th space. On palpation the apex beat can be felt feebly in the 5th space about ½" internal to midclavicular line. The heart sounds are audible but feeble in all the areas. No murmurs heard.

Lungs: Fine crepitations are heard over the bases more on the left side. The breath sounds over the left base, in the axillary region and in front are very feeble and distant.

Abdomen: Liver—lower border 3 fingers below the costal margin, soft in consistency. Spleen palpable. Small amount of free fluid also present in the abdomen.

### Progress of the Case

16-4-1949—80 c.c. of hæmorrhagic fluid aspirated from the pericardial sac.

20-4-1949—160 c.c. of hæmorrhagic fluid aspirated from the pericardial sac. Proteins 2·8 per cent. Globulin +. Smear packed with erythrocytes, 7-8 lymphocytes and 1-2 polymorphs per field No organisms including acid-fast bacilli seen. No growth of pyogenic organisms could be obtained.

22-4-1949—Temperature ranges from 99-102°F. Sterptomycin ½ gm. twice a day was started. Cardiac dullness diminished, patient feeling better. Less dyspnœa.

24-4-1949—Temperature has come down to 100°F, the patient feeling better; pulsus paradoxus present.

26-4-1949—Temperature went upto 103°F, the area of cardiac dullness was found to be decreasing; pulsus paradoxus was present.

28-4-1949—Liver less tender and much diminished in extent. Pulsus paradoxus present.

30-4-1949—Condition improving. Findings same.

2-5-1949—Condition same. Temperature 99°-101°F. Pericardial friction heard over mitral area. Liver much diminished but still tender.

5-5-1949—Feeling better. Findings same.

7-5-1949—Condition same. Friction heard over a bigger area. Fluid seems to have increased on the right side.

10-5-1949—Improving satisfactorily. Temperature 99-101°F. Friction heard all over the præcordial area. Area of cardiac dullness much decreased. X-ray revealed fluid on the right side of chest. Costophrenic angle of the left side also appears obliterated.

12-5-1949—Temperature 98-102°F. Breath sounds and vocal fremitus absent below the level of the 4th rib on the right side.

13-5-1949—1200 c.c. of hæmorrhagic fluid removed from the pleural cavity. One hour after this the patient became suddenly dyspnœic. Put on oxygen and coramine the cyanosis disappeared in about 3 hours. 25 c.c. of 25 per cent glucose was given at 8-30 A.M.

14-5-1949—Feeling better. Complains of pain in the inframammary region. E. S. R. 55 mm. 1st hour, 90 mm. 2nd hour. Temperature, 99-102°F.

17-5-1949—Sputum no acid fast bacilli seen.

22-5-1949—Fluid in the chest has decreased. When lying down the fluid level appears to be at the level of the 5th rib on the back. Pericardial rub has disappeared. The apex on percussion and palpation coincides. Temperature 99-100°F.

28-5-1949—Screening chest. The fluid level on the right side is at the level of the 4th rib anteriorly in the anterior axillary line. Heart shadow is enlarged.

30-5-1949—Temperature 98-100°F, patient feeling better, fluid level same.

2-6-1949—Pulsus paradoxus more marked. Temperature 102°F. Patient complains of feeling like vomiting. Clinically no fluid in the pericardium. E. S. R. 56 mm. 1st hour.

3-6-1949—No growth of tubercle bacilli obtained after 6 weeks incubation from pericardial fluid.

4-6-1949—Temperature 100°F. Uncomfortable to some extent due to fear. Complains of pain in the back. Pulsus paradoxus marked. Fluid level 5th rib anteriorly.

8-6-1949—Temperature 99°F. Feeling a bit better.

12-6-1949—Screening chest. The capacity in the lower zone of the right side does not seem to be due to fluid. It appears to be a pulmonary lesion.

21-6-1949—Temperature 98-99°F. Pulsus paradoxus present. Liver enlargement same as before. Dullness at right base.

27-6-1949—Total white cell count was 11,000 with a differential count of poly 72 per cent and lympho 28 per cent. Temperature normal. Feeling better.

30-6-1949—Growth of tubercle bacilli obtained from the pleural fluid.

12-7-1949—Temperature normal. Patient feeling allright. X-ray and screening of the chest revealed fluid at the right costophrenic angle. Heart area enlarged. But there is no clinical evidence of fluid in the pericardium.

20-7-1949—Patient discharged from the hospital and advised to attend the chest clinic for follow-up.

Patient has been at the clinic several times after that. There is no evidence of fluid in any of the above cavities. His weight has increased gradually and he feels fit. The last letter received from the patient dated 24-2-1951 intimates that he feels perfectly fit and is doing his normal work.

### Discussion

That the polyserositis was definitely of tubercular origin has been shown by the organism isolated from the pleural fluid. Although when he came under observation, he had fluid in the pericardium and abdomen, he developed a large quantity of fluid in the right side of the chest and a very small quantity in the left. He was not given any P. A. S. and was treated with 90 gm. of streptomycin. He has been followed till 24-2-1951 and has had no setback. A few cases of success with streptomycin in treating pericarditis have been found in the literature and due to rarity of the condition this case is reported.

Although he was on streptomycin, he developed a large quantity of fluid 21 days after the treatment was started and despite the administration of streptomycin for 21 days before the specimen of fluid was sent for culture, a positive growth of tubercle bacilli was obtained. It took 55 days for the temperature to be controlled and this is very much against the view that the course of the drug should not be more than 45 days when the chances of drug-fastness increase. In this case it is through perseverance with the drug that success appears to have been achieved.

### Summary

1. A case of polyserositis of tubercular origin has been described.

2. He developed pleural fluid during the course of treatment.

3. A positive culture was obtained from the pleural fluid.

4. It took 55 days for the temperature to be controlled.

## A CASE OF ANTEPARTUM STRANGULATION OF A POSTMATURE FOETUS

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*Senior House Surgeon, Eden Hospital, Medical College, Calcutta.*

The umbilical cord is frequently found, during labour, to have coiled itself several turns round the neck of the fœtus, and this is recognised as an uncommon cause of intrapartum fœtal asphyxia. De Lee's book gives a nice picture. A case was recently seen by us and is reported here.

Mrs. A. B. aged 34, was admitted to the hospital on 6th May, '51, seven days overdue and complaining of pain in the abdomen (vague, all over) and absence of fœtal movements for the last two days. She had 3 previous pregnancies all of which had ended in full-time natural and spontaneous live births, the last one 5 years back. The present pregnancy was however not quite normal. The patient had vaginal bleeding in the early months, diagnosed as a case of threatened abortion, treated with sedatives and confinement to bed. The patient carried on with the pregnancy, with increasing enlargement of the uterus, in spite of similar attacks of slight pelvic pain and vaginal bleeding, repeated off and on. But these did not worry the patient or her doctor very much, as the fœtus was being regularly followed "alive and kicking". She was given a dose of castor oil and an enema on the calculated expected date of confinement—without any result. Three days before her admission (*i.e.*, when 4 days overdue) the patient felt violent fœtal movements for about half an hour, after which she did not feel any movement of the fœtus. Examination showed a term pregnancy, absent fœtal movements and f.h.s., vertex presenting, and a soft fœtal head without clear-cut outlines but no egg-shell-crackling on vaginal exploration. The patient went in labour spontaneously in about 10 hours after admission and was confined unaided within 2 hours of a stillborn macerated fœtus strangled by three tight turns of the cord round its neck, the cord having a total length of 24 inches.

It is difficult to explain the cause of the repeated attacks of pelvic pain and vaginal bleeding in the early months of pregnancy; the placenta was not implanted low nor did it show multiple small old infarcts as might be expected. The fœtus had obviously died after the violent movements 4 days before its birth, and directly due to tight coils of the cord stopping the circulation through it. There were no recent big placental infarcts nor any retroplacental hæmatoma; hence it seems unlikely that postmaturity (only 7 days) as such, or the attempt at medicinal induction of labour, was responsible for the antepartum intrauterine fœtal death which was perhaps aided by the somewhat extra length of the umbilical cord.

My grateful thanks are due to the Superintendent of our hospitals and my chief Prof. M. N. Sarkar for their kind permission to use the hospital records and to Dr. R. Sinha for his kind help and guidance.

JOURNAL OF THE

INDIAN MEDICAL ASSOCIATION

CALCUTTA, NOVEMBER, 1951

## SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION

The surgical measures in the treatment of essential hypertension have been employed for nearly twenty years. The phase of trial and error is over, and it should now be possible critically to assess its exact position, and to ascertain its value in the relief of this condition.

The modern operation is a combined supra- and infra-diaphragmatic extrapleural and extraperitoneal procedure, and aims at extensive sympathetic ganglionectomy from the fourth dorsal to the second lumbar segments, together with splanchnicectomy. The operation is bilateral, and is usually done in two stages at an interval of a fortnight. This is a combination of Smithwick's[1] and Poppen's[2] operations, and has been evolved through successive stages of improvements, since the original subdiaphragmatic approach was first described by Adson, Smith and Brown.[3] The mortality from this operation in skilled hands and after careful selection of cases should be very small. Learmonth[4] places it at less than one per cent. In Mcgregor's[5] series, there were two deaths. In the cases reported from the British post-graduate school the mortality was even less.[6]

If the aim is to obtain the maximum benefit from the operation, and not just a temporary palliation, cases should be selected with meticulous care. The criteria by which such selections are made are the age of the individual, the resting blood pressure, especially the diastolic pressure, its response to sedation, the renal function, the state of the cardiovascular system, eye-ground changes and history of cerebrovascular episodes. An ideal case for this operation should be less than 50 years of age, the blood pressure should range between 170 and 105 mm. Hg. and it should respond well to sodium amytal, and to rest and sleep. The urea clearance should be over 40 per cent, the blood urea less than 40 mgm. per 100 ml. The specific gravity of urine should not be less than 1020. The cardiac function should be well compensated. The extent of eye-ground changes should not be beyond group II described by Keith, Wanger and Barker[7] and if there has been any cerebro-vascular episodes in the past, the patient should at least have completely recovered from it. These are exacting standards, and if they are to be tresspassed, as they sometimes are, this should be done only by an expert. For the occasional sympathectomist, it is best to adhere to these rigid limits in order to obtain satisfactory results.

To the common man the succes of any operative procedure, or indeed any method of treatment, is judged not by statistical data or by laboratory tests, but by the individual happiness that results from such treatment. On this standard, there is much to be said for this "great physiological experiment". There is no doubt that, in a large proportion of selected cases it brings about marked symptomatic relief, improves the working power and enhances the expectancy of life.

Smithwick[8] in 1948 reported on 256 patients followed up from 5 to 9½ years after the operation. This is the largest series and the longest period of follow-up recorded by any author after this operation. He analysed his cases critically from several aspects. The overall mortality of his cases during this long interval was 31 per cent. The renal function improved in 28·9 per cent, deteriorated in 9·8 per cent and remained unchanged after the operation in 9·8 per cent of his cases. Examination of eye-grounds revealed that as a result of the operation the condition improved in 40·7 per cent, there was no change in 39·2 per cent and it was worse in 20·1 per cent of cases. Electrocardiography showed that the cardiac condition improved in 42·3 per cent, remained stationary in 48·8 per cent, while in only 8·9 per cent it became worse. The operation succeeded in markedly lowering the blood pressure in 21 per cent of cases. The reduction of blood pressure was moderate in 13 per cent, slight in 13 per cent, not significant in 36 per cent. In 17 per cent the hypertension increased in spite of the operation. The survival curve or the life-expectancy was very interesting. Plotted on the same basis as the curves prepared by Keith, Wagner and Barker (*loc. cit.*) in medically treated, or untreated cases, this showed considerable improvement in all the four groups. The differences were statistically significant especially in groups 2, 3 and 4.

Similar statistical data are also available from the reports of other observers. Mcgregor (*loc. cit.*) analysed 50 of his personal cases followed up for an average period of 2 years. Eighty per cent of his patients had a worthwhile fall in the resting diastolic pressure, and 90 per cent had relief of symptoms. The damage done to the eye-grounds, myocardium and kid-

[1] *Surgery*, **7**, 1, 1940 *& Arch. Surg.*, **49**, 6, 1944.
[2] *Jour. Am. Med. Assoc.*, **134**, 4, 1947.
[3] *Surg. Gyna. & Obst.*, **62**, 314, 1936.
[4] *British Surgical Practice, Butterworth, London*, **5**, 70, 1948.
[5] *Brit. Jour. Surg.*, **36**, 281, 1948.
[6] *Brit. Med. Jour.*, **1**, 665, 1951.
[7] *Am. J. Med. Sci.*, **197**, 332, 1939.
[8] *Brit. Med. Jour.*, **2**, 237, 1948.

neys, in his opinion, were remedied by the operation in an appreciable percentage of cases.

The recent report from the British post-graduate medical school (Chris[8]) also corroborates many of the above conclusions.

In spite of the beneficial effects of this operation, some undesirable post-operative symptoms sometimes cause considerable annoyance. But these are neither serious nor affect the ultimate prognosis. The post-operative pain may trouble the patient for many months. Giddiness or transient "black-out" when getting up suddenly may occur and sometimes Raynaud's phenomenon affecting the hands or angina pectoris in arteriosclerotic subjects may incapacitate the patient for a long time. In many cases, convalescence is unduly prolonged and the individual becomes lethargic, apathetic and sometimes psychotic. It is therefore, essential that a possibility of these symptoms should be considered when planning an operation in an individual case.

In spite of a fairly exhaustive survey of the subject in American and British literature, there has been a marked paucity of publications in the medical journals of India concerning this operation. It is probable that Indian surgeons have not yet taken to this operation in any large numbers. The cause of this is difficult to ascertain. Whether this attitude has been due to the aversion of the surgeons themselves, or to the lack of appreciation of the benefits of this operation by the general physicians, on whom the responsibility of referring suitable cases to the surgeons primarily rests, is difficult to determine. We therefore, welcome the paper by Chandy[9] which appears in the present issue of the Journal and which is a well documented summary of the physiology, anatomy and the different techniques of this operation. We would however have liked to see the results of this operation in the cases treated by Dr. Chandy. Two years ago Cooper[10] also reviewed this problem but his paper also similarly lacks in reports on personal cases. However, the Association of Surgeons of India have arranged at their annual conference in 1952, a discussion on the surgical treatment of hypertension. It is hoped that at that time the results of this treatment obtained by the surgeons in this country will be available for study.

## PREVENTION OF FOOD ADULTERATION

India is faced with an acute food crisis. There is a definite overall shortage. The little there is to go round is of poor quality and is frequently adulterated. Whatever the people get must be wholesome and free from adulteration. The medical profession are only too well aware of the effects of adulterated food on the health of the people. The consumption of such foodstuffs brings in its wake a number of maladies, which make one temporarily, if not permanently, incapacitated for work.

We therefore welcome the announcement the Hon'ble Health Minister recently made in Parliament while replying to a question. According to the Rajkumari's statement, a comprehensive amendment to the existing Food Adulteration Act has been taken up in right earnest. The recommendations of the various States are being scrutinized by legislative experts and a Bill is to be introduced in Parliament very soon.

This is a move in the right direction. The question of introducing measures aimed at improving the machinery for effective control over adulterators and the adulteration of foodstuffs, rampant of late, is urgent and brooks no delay.

The extant law has many loopholes. It is the small fry the retailer who generally gets punished, while the bigger culprit the wholesaler usually manages to evade its meshes. Many will recall in this connection the ravages caused in Eastern India by argemone-adulterated mustard oil some two years ago. The adulteration took place in one province, the oil was expressed and consumed largely in other provinces. Provincial Governments punished some of the dealers but could not touch the source being outside their own jurisdiction. Such an anomalous state of affairs resulted from defects in the law. The question of State jurisdiction definitely hampers the rounding up of large-scale inter-State operators. Those who are hauled up seldom get adequate punishment. The punishment must suit the crime and we can hardly think of a more heinous crime than mucking up our food. We seriously ask the Government to consider some deterrent punishments; fines and imprisonment are not sufficient deterrent. The present procedure of sample collection, analysis and sanction by local bodies is cumbersome and long drawn-out.

We can ill afford to subject ourselves to the whims of the criminally greedy and dishonest dealers who make their pile by depriving the nation of its vital needs for the growth and development of the people. The food that is served to the people must be dependable with all its calories, minerals and vitamins assured or malnutrition and its concomitant factors are sure to develop.

It is on these premises that we welcome the Government of India's decision to proceed with the amendment of the Food Adulteration Act. Let us hope, out of the labours of Parliament will emerge an enactment that will assure our people wholesome and unadulterated foodstuffs, so vital for the regeneration of national health.

[9] *Jour. Ind. Med. Assoc.*, **20**, 1951.
[10] *Calcutta Med. Jour.*, **45**, 337, 1948.

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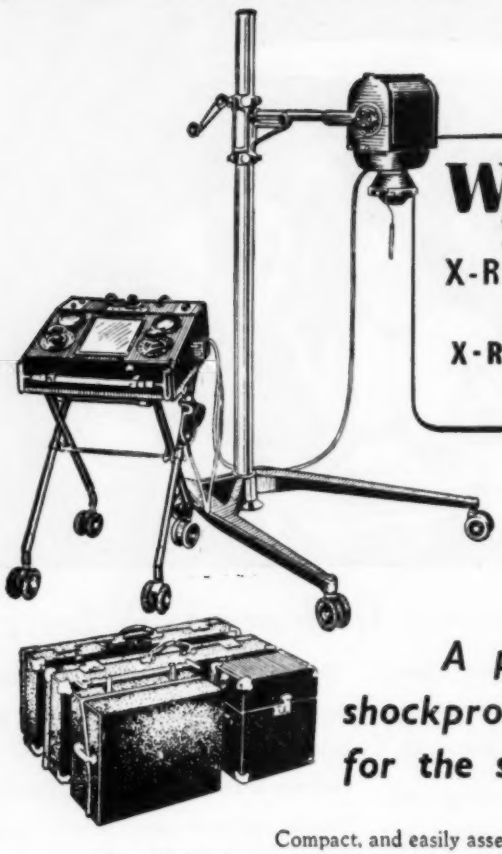

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# CURRENT MEDICAL LITERATURE

### Congestive Heart Failure and Hyponatraemia

Citron and others (*Ann. Int. Med.*, **34**:872, 1951) observe that the administration of mercurial diuretics to patients whose sodium chloride intake is restricted may result in hypotonicity of the body fluids.

Usually the electrolyte deficiency does not manifest itself clinically, but occasionally anorexia, apathy, nausea, muscular pains, psychosis and coma are observed.

Oliguria and azotæmia sometimes occur and may give one the erroneous impression that therapy (*i.e.*, salt restriction and mercurialization) has not been vigorous enough. At such a point more rigid salt restriction and more frequent administration of mercurials are fraught with danger.

The symptoms and signs of salt depletion are usually alleviated by the administration of adequate amounts of hypertonic sodium chloride solution.

### Bronchitis and Bronchiolitis in Pulmonary Disease

Cardon and others (*Ann. Int. Med.*, **34**:588, 1951) write that an episode of acute bronchitis ingrafted on chronic asthmatic bronchitis and emphysema, pulmonary fibrosis or bronchiectasis may so obstruct the bronchi that an acute respiratory emergency and death by suffocation may be precipitated.

This disease is often misdiagnosed as acute left ventricular failure or pneumonia. The consequent delay in proper therapy contributes to the mortality from asphyxia.

The essential therapeutic measures are: repeated aspiration of the tracheobronchial tree, administration of oxygen in adequate concentration, and the use of bronchodilator, vasoconstrictor, antibiotic and chemotherapeutic agents. Supportive and stimulant measures and the prevention and treatment of complications are important adjuvants.

Even a mild upper respiratory infection in patients with chronic pulmonary disease must be treated seriously from its very incipience. If progress is unfavourable, early hospitalization for constant observation and early drastic treatment is imperative.

Residence in a warm, dry climate during the late autumn, winter and early spring is valuable in the prophylaxis of this serious asphyxial disease.

### Portal Cirrhosis with Ascites

Ricketts (*Ann. Int. Med.*, **34**:37, 1951) writes that the medical management of patients with or without ascites is similar except for sodium restriction in patients with ascites. Fluid retention can be controlled by restricting the intake of sodium. Dietary management with a high protein, high carbohydrate and high caloric intake, plus additional choline chloride, tends to improve nutrition and hepatic parenchymal regeneration, to bring about sustained rise in plasma albumin, and thus to eliminate œdema and ascites. This effect is obtained after continuous treatment for several months. Plasma albumin values apparently can be restored only after repletion of tissue protein. Repeated paracenteses should be avoided, since they result in a marked loss of protein. No tendency to recurrence of ascites is seen in uncomplicated cases maintained on an adequate nutrition.

The immediate prognosis of jaundiced patients with ascites depends on the severity of the parenchymal failure. The ultimate prognosis of patients who have recovered from œdema and ascites remains guarded, and is determined to a great extent by the incidence of complications, the most frequent of which is bleeding from varicose veins of the œsophagus.

### Brewer's Yeast in Hepatitis

Davis (*M. J. Australia*, **2**:790, 1950) evaluates the results of therapy in 126 patients with early acute or subacute hepatitis. Late cases of advanced cirrhosis of the liver were not included in this series. When a patient came under observation, liver function tests (galactose tolerance, serum cholesterol estimation and hippuric acid excretion) were carried out, and these were repeated at intervals of three to six months throughout the treatment. In the 19 severe cases the patients were put to bed for a period of four to six weeks. All other patients were allowed to carry on their duties without interference. All were given a diet of high caloric content consisting of 140 gm. of protein, 350 to 400 gm. of carbohydrate and 70 gm. of fat. To this diet was added vitamin B complex consisting of a daily dosage of 30 mgm. of $B_1$, 5 mgm. of riboflavin and 25 mgm. of niacin. Neither choline nor methionine was included in the diet. All patients were given 12 gm. of dehydrated brewer's yeast daily. This was given during each meal, dissolved in barley water, milk or fruit juice. With this treatment, 29 patients were cured, 40 were improved, 52 were relieved, four obtained no relief and one died. The author feels that dehydrated brewer's yeast containing a high percentage of vitamin B has definite therapeutic value.

### Gastrointestinal Syndrome of Reducible Hernia

Gauss (*Am. J. Digest. Dis.*, **18**:146, 1951) gives in the following lines the summary of his observations:

Reducible hernia may give rise to a gastrointestinal syndrome.

The digestive syndrome may simulate peptic ulcer, the gastric irritation syndrome, the irritable colon syndrome or variants of these.

The patient frequently considers the digestive symptoms his primary problem.

The gastrointestinal syndrome is reflex in origin and functional in character.

The referred symptoms are mediated through the sympathetic nerves. Irritation of the contents of the hernial sac initiates an irritation of the bowel, which can refer its symptoms to remote parts of the abdomen.

Treatment of the referred gastrointestinal syndrome consists in correcting the hernia surgically; following which the digestive symptoms usually subside spontaneously.

Patients may have co-existing hernia and primary digestive problems. When the digestive problems are present, they require appropriate treatment.

The abdomen has been called the spokesman of the body. It tells trouble, but it does not locate it. The task of locating the source of the symptoms is the responsibility of the physician.

### Terramycin on Intestinal Flora

Dearing and Needham (*Proc. Staff Meet. Mayo Clin.*, **26**:49, 1951) write that the studies among 68 patients who had various types of intestinal lesions requiring surgical treatment indicate that as an antibacterial agent on intestinal flora terramycin is as effective as aureomycin.

For preoperative intestinal antisepsis the authors recommend that terramycin be employed orally in the same doses employed for aureomycin; that is, 750 mgm. given four times daily for three to three and a half days.

Cultures of the fæces from patients who received terramycin showed that certain organisms were relatively resistant to the drug. Among these were the genera Proteus and Pseudomonas, certain coagulase-negative micrococci, and occasional strains of Aerobacter aerogenes and Streptococcus fæcalis. Yeasts were very resistant to terramycin and tended, when present in the stools, to increase in numbers.

Nausea and vomiting were the only significant symptoms of toxicity produced among the patients who received terramycin. The frequency of occurrence and the intensity of the nausea could be reduced to a minimum by feeding the patients when the terramycin was administered.

### Aureomycin in Abacterial Pyuria

Baurys (*J. A. M. A.*, **145**:403, 1951) in reviewing two cases of abacterial pyuria treated with aureomycin observes that the response to therapy was dramatic. Aureomycin appears to be far superior to other therapeutic agents for this urinary tract disease.

### Tetanus Prophylaxis with Penicillin-Procaine

Taylor and Novak (*Ann. Surg.*, **133**:44, 1951, Ref. *J. A. M. A.*, **145**:1092, 1951) suggest for prevention of the development of tetanus in contaminated wounds the local use of penicillin rather than antitoxin, which only neutralizes toxin after it forms without eliminating the source. To demonstrate the effectiveness of this procedure they inoculated viable spores of Clostridium tetani into devitalized tissues of several groups of mice and followed this with slowly absorbed penicillin injected locally in one group and systemically in another. For comparison other groups received tetanus antitoxin locally and systemically, alone and in combination with penicillin. A control group received no prophylaxis. The mortality in the control group was 100 per cent. The mortality in the mice receiving penicillin locally within six hours after infection in doses equivalent to 600,000 to 1,200,000 units in man was only 5 to 10 per cent. When penicillin was injected systemically rather than into the infected area, or was administered 24 hours after infection, it did not protect the animals from death. Antitoxin alone injected locally or systemically at any time within 24 hours after infection in amounts equivalent to 1,600 to 100,000 units in man provided no protection. A combination of penicillin and antitoxin produced the same results as penicillin alone, except that when injected 24 hours after infection the combination reduced the mortality 35 to 58 per cent, whereas either agent alone gave no protection. It is assumed that penicillin prophylaxis will be equally effective in man.

### Changes in Blood after Operation

Wilkinson and others (*Lancet*, **1**:315, 1951) give in the following lines the summary of their observations on changes in blood chemistry after surgical operations:

Changes in the composition and volume of the blood after severe surgical operations followed a general pattern which was independent of the type of operation or the method of postoperative feeding.

In all the patients there was a sudden fall in the plasma-protein, in particular the albumin concentration. In most cases the globulin concentration gradually rose to values well above the preoperative levels.

Increases in amount of non-protein (urea) nitrogen were observed in some patients.

Hæmoconcentration on the day after operation followed by hæmodilution, was observed in all patients. Changes in hæmatocrit values were accompanied by corresponding changes in red-cell count.

An immediate reduction in total blood-chloride with an inconstant reduction in plasma-chloride was followed in most patients by a rise in total blood-chloride. There were corresponding changes in the serum-sodium. There was some evidence that electrolyte ions were transferred from the plasma to the red cells.

On the day after operation there was a significant reduction in plasma and blood volume, and in the total amounts of proteins and sodium circulating in the plasma, but no significant change in the "thiocyanate space."

The relation of the blood changes observed to the periods of salt retention and protein katabolism after operation is discussed.

### Potassium Deficiency in Pyloric Stenosis

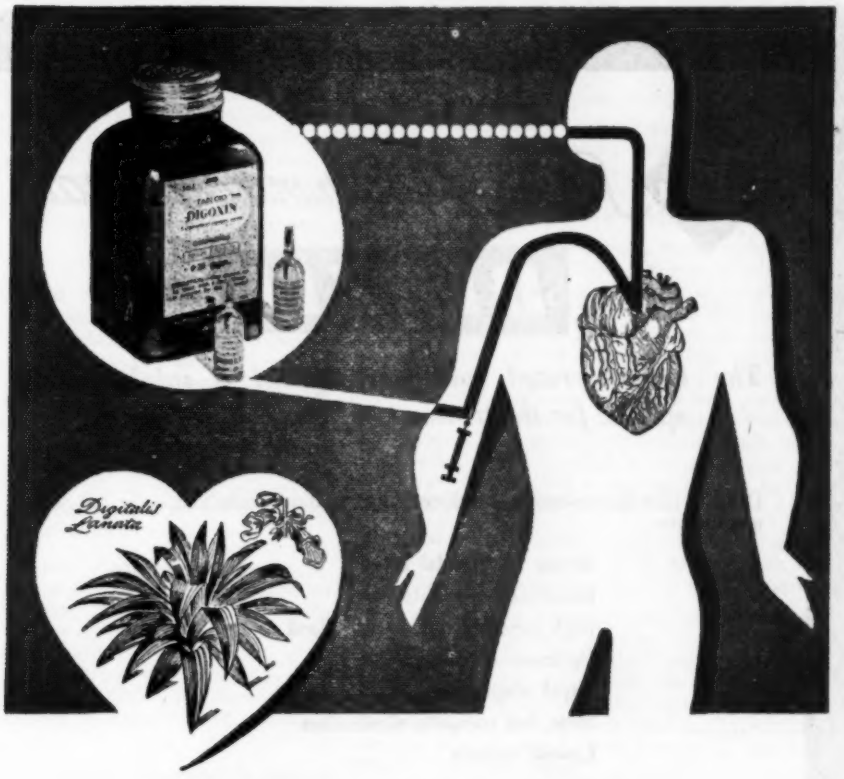
Hawkin and others (*Lancet*, **1**:318, 1951) write:

A patient with advanced pyloric stenosis suffering from salt-depletion and extrarenal uræmia was only temporarily benefited by glucose-saline infusions. His apathy, muscular atonia, bradycardia, and shallow respirations pointed to potassium deficiency, which was confirmed by the electrocardiographic findings and was not excluded by a serum-potassium level only on the low side of normal.

Aetiological factors, such as undernutrition, vomiting, and dehydration are discussed, and the dangers of infusing such a patient with sodium chloride are indicated.

Early recognition of hypokalæmia is of great importance in such cases which do not respond to the usual treatment of salt-depletion and uræmia.

In the present case, the subcutaneous infusion of potassium chloride resulted in dramatic improvement in the symptoms and reversal of the electrocardiographic abnormalities. For

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a time the symptoms of hypokalæmia returned if the infusion was stopped, but ultimately the patient fully recovered.

### Danger of Penicillin Therapy in Gonorrhœa

Reekie (*Lancet*, **1**:327, 1951) writes that unexplained fever and malaise occurring shortly after penicillin therapy for gonococcal urethritis strongly suggest co-existing syphilis, and the symptoms and signs of the oncoming syphilis may be suppressed, masked, or delayed. Patients displaying such reactions should be subject to very close scrutiny, both clinical and serological, for six months from the start of treatment.

### Jaundice and Pregnancy

Jawert and Morrison (*Texas State J. Med.*, **47**:137, 1951) write that the two types of jaundice in pregnancy are (1) the icteric or clinical variety, and (2) the anicteric or subclinical type. Arbitrarily, an icterus index of 15 units has been used to distinguish both forms. Jaundice and acute yellow atrophy of the liver have many different contributing causes; both are rare complications of pregnancy, jaundice having an incidence of 1:1,400 and acute yellow atrophy, 1:10,000 patients. Among 51 patients with jaundice, 32 had medical conditions and 9 had lesions requiring surgery; two obstetric conditions, hyperemesis gravidarum and acute yellow atrophy of the liver, accounted for 10 cases.

When a patient develops hyperemesis and epigastric discomfort, she should be promptly admitted to the hospital for correction of the electrolytic balance and other metabolic derangements. Under such a regimen no case of acute yellow atrophy has been observed clinically in 28,000 patients at the New York Hospital since 1943, during a period when infectious hepatitis was rather prevalent. At times a paraœsophageal hernia was noted to be the cause of vomiting in the third trimester.

A careful differential diagnosis is necessary in order to institute proper medical, surgical and obstetric treatment. Gallstones were treated without regard for the pregnancy. Only 1 patient with hepatitis had an induction of labour. Four of the 7 patients with acute yellow atrophy had the labour terminated by various methods, with 1 death.

Since acute yellow atrophy has many ætiologic causes, it may be well to attach a qualifying phrase such as acute yellow atrophy "of pregnancy", "of hepatitis", "of chloroform," and "of transfusions" rather than to discontinue its use as a diagnosis. The term is too deeply ensconced in the obstetric literature to do otherwise.

Obstetric conditions causing an elevated icterus index level (from 6 to 15 units) were premature separation of the placenta, missed abortion, and erythroblastosis fœtalis. It is likely that patients with other conditions such as threatened abortion and ectopic pregnancy also have an elevated icterus index level. It seems significant that only 1 patient of the 51 with jaundice in pregnancy had pre-eclampsia and this was associated with hepatitis.

The routine use of the icterus index test and a urine test for bile provides an excellent opportunity for the study of obstetric patients with regard to anicteric and icteric jaundice. Such a program will increase the total number of cases for study and consequently increase our knowledge of the causes of jaundice in pregnancy.

### Intravenous Iron in Anæmia of Pregnancy

Scott and Govan (*Lancet*, **1**:367, 1951) write:

The patients formed three main groups: 78 antenatal cases of uncomplicated iron-deficiency anæmia; 59 cases of puerperal anæmia; and 13 antenatal cases of anæmia resistant to iron by mouth. All the antenatal patients were in the last trimester of pregnancy when intravenous iron was administered.

*Dosage*—In general the following regime has been adopted: On the first day the equivalent of 30 mgm. of elemental iron is given, on the second day 60 mgm., and on the third day 100 mgm. Thereafter 100 mgm. is given daily until the end of the first week. During the second week 100 mgm. is given every second day, so that by the end of this week 790 mgm. of elemental iron has been given. This is sufficient to raise the "moderate" cases above the anæmia level, and they may then be given iron by mouth if necessary. Patients with severe anæmia may continue with intravenous iron 100 mgm. every second day in subsequent weeks, or the dose may be reduced to 100 mgm. once a week according to the severity of the anæmia.

It has not been possible for the authors to use larger doses than 100 mgm. of any of the three products, because of the frequency of reactions when that figure is exceeded.

*Reactions*—Minor reactions, such as pain in the arm, are fairly common, occurring in about 20 per cent of cases. The most alarming reaction in pregnant women only occurs in about 1 or 2 per cent of cases, and takes the form of a vasovasal attack. It occurred in a few patients to whom it was attempted to give more than the usual dose of 100 mgm. of elemental iron. It may be seen, however, in some patients even with the usual dosage.

*Results*—The results of intravenous iron therapy in 150 cases of anæmia of pregnancy and the puerperium are recorded:

During the antenatal period most of the cases responded immediately, and with the dosage used there was an average increase of 1·5 gm. of hæmoglobin per 100 ml. in a week.

About a third of the patients showed a delay in response until the second week of treatment. This delay seemed to be related to the availability of protein.

During the later months of pregnancy the highest response increase of hæmoglobin was 0·4 gm. per 100 mgm. of elemental iron.

Treatment with intravenous iron at this period induced a temporary hæmodilution lasting three days in moderate anæmia and four or more days in severe anæmia.

During the puerperium the average response to intravenous iron almost reached the calculated level of 4 per cent per 100 mgm. of elemental iron. In a few cases this figure might be exceeded, probably owing to the normal hæmoconcentration which begins usually about the third day after delivery.

Response was delayed in the puerperium in infections, in systemic diseases, and in patients who had suffered shock at delivery.

Iron-deficiency anæmia which had proved resistant to iron by mouth improved rapidly under intravenous therapy. The possible causes of resistance to oral iron are discussed.

It is recommended that in the treatment of anæmia of pregnancy no single dose should exceed 100 mg. of elemental iron. Severe reactions tend to develop when higher doses than this are given.

### Vitamin K and Haemorrhagic Disease

Hay and others (*Lancet*, **1**:423, 1951) write that vitamin K was administered to 4602 pregnant women before delivery. There were 12,131 controls. Hæmorrhagic disease was observed in 11 of the newborn infants of the vitamin K group (1 in 418) and in 23 of the controls (1 in 527).

### Diphtheria in Calcutta

Das and Ghosal (*Lancet*, **1**:1410, 1951) write:

115 strains of Corynebacterium diphtheriæ isolated from diphtheria cases in Calcutta were found to be mitis only.

Excluding cases admitted after the fifth day, the death-rate was 15·3 per cent.

Laryngeal obstruction (48·6 per cent) was the most prominent feature. Cardiovascular and neurological involvement was found in 29·6 per cent and 8·6 per cent respectively. Death in the early stage was caused by extension of the membrane, and later by toxæmia.

The disease caused by the mitis type of *C. diphtheria* in Calcutta seems to be much more serious than the disease it causes in England.

### Cyclical Vomiting

Hamilton (*Lancet*, **1**:330, 1951) at the section of Pediatrics of the Royal Society of Medicine said that cyclical vomiting was first described by Gee under the name "fitful" or "recurrent" vomiting. Attacks started at the age of 3 years or less, and they usually ceased spontaneously around the age of 12. The patient was likely to be thin and nervous; often he was an only child, and commonly there was a family history of similar attacks, of travel-sickness, or of migraine. Attacks might be precipitated by shock, strain, excitement, infection, or dietary indiscretion; and a prodromal period of listlessness could often be recognised. After the first vomit, which might contain food taken many hours before, nothing taken by mouth could be retained, and the vomit consisted of gastric juice containing mucus, bile, and possibly blood. Associated symptoms were nausea, abdominal pain, headache, and giddiness; some of these manifestations might be more conspicuous than the vomiting, and occasionally there were convulsions. The patient might be pale and febrile and either drowsy or restless. The blood-pressure was sometimes raised, and the urine contained acetone. A severe attack might end fatally, but usually recovery took place quite suddenly after two or three days.

The abdomen was lax and easy to palpate, so confusion with acute inflammatory abdominal diseases did not usually arise; but the condition was often difficult to diagnose from recurrent intestinal obstruction, intracranial tumour, and diabetic coma (glycosuria often being present). The first attack was, of course, always difficult to diagnose. Recurrent vomiting was closely connected with recurrent pyrexia without obvious cause, attacks of which might alternate with vomiting, and also with recurrent headache; but no relation with epilepsy or with allergy had been demonstrated.

Prof. Stanley Graham said that the condition could be defined as "periodic attacks of vomiting associated with marked ketosis." The attacks were sometimes called "acidosis", but this was a bad term because acidosis did not necessarily accompany the ketosis; and indeed there might be an alkalosis. In hospital attacks did not occur spontaneously, and could not be induced by giving a ketogenic diet or ammonium chloride. Professor Graham disliked the term "cyclical", because the attacks do not have any definite rhythm.

The basis of the condition seemed to be an abnormal production of ketone bodies; and possibly this represented an abnormal response by the liver to some form of stress, rather than a disease entity. Turning to treatment, Professor Graham emphasised the importance of treating the child as a whole; administration of extra carbohydrate between attacks was probably beneficial, but the value of fat reduction was doubtful. There was no theoretical basis for the regular administration of alkalis.

Dr. Desmond O'Neill spoke on the psychiatric aspect of abdominal pain. He described a study made at Guy's Hospital by Dr. R. C. MacKeith and himself on a consecutive series of children attending the outpatient pædiatric clinic with recurrent abdominal pain as a presenting symptom, for which no physical cause could be found. In several of these children vomiting occurred in some of the attacks. The purpose of this study was to determine whether the abdominal pain could be regarded as an adaptive disorder that is, whether it was related to emotional tension from maladaptation to stresses which the child had to meet. It was found that emotional tension was, in fact, the principal determinant of the pain in 15 out of 25 children, and that it was a contributory factor in another 6. Of this group of 21, 17 had other tension signs, such as headache, poor sleep, tics, and behaviour disorders. In 14 there was sleep, tics, and behaviour disorders. In 14 there was a history of nervous disturbance or stress disorder in one or both parents.

Anxiety and fear were much the most common emotional states associated with pain; fear of the teacher at school or of being bullied was often found. Once the attacks had been set in motion in this way, a complaint of abdominal pain might be made in order, consciously or unconsciously, to escape from an unpleasant situation. The effect of simple psychotherapy, in which play therapy had an important part, was striking, reduction or disappearance of the pain being achieved in a high proportion of cases. In some children under treatment the pain was replaced by another symptom, such as screaming attacks; and this had in turn to be treated.

Dr. R. E. Smith, whose paper was read in his absence by Dr. D. V. Hubble, dealt with the prognosis and sequelæ of recurrent vomiting, basing his conclusions on a study of 700 boys between the ages of 13 and 18 at Rugby School. Of those who had had attacks in childhood nearly a quarter continued to do so in adolescence, but there was a well-marked decline around 15-16 years of age. Acute appendicitis

might be overlooked in a boy subject to these attacks, but otherwise they were not serious.

Dr. Hubble also described the findings in a fatal case of his own, in which there was hypertrophy of the pylorus. He suggested that during life there might have been pylorospasm and reversed peristalsis, and he thought that a similar mechanism might account for the vomiting of migraine. He had found ergotamine tartrate of value in recurrent vomiting as well as in migraine.

Dr. R. C. MacKeith remarked on the interval of up to 48 hours that often elapsed between the exciting cause and the onset of the attack.

Dr. J. P. M. Tizard referred to some organic causes of recurrent vomiting, such as tonsillitis, pyelitis, œsophageal stenosis, and intestinal obstruction; and Dr. H. R. Jolly mentioned lead poisoning.

Dr. J. J. Kempton observed that the subjects of recurrent vomiting are often in a state of mild chronic ill health, correction of which suffices to bring about improvement.

Prof. A. A. Moncrieff emphasised the importance of adequate rest for these children, and the value in many cases, of a regular sedative. The infections precipitating attacks might be very slight, and detectable only by careful examination. Professor Moncrieff also spoke of the need for a control series in evaluating the role of emotional factors in causing symptoms.

Dr. W. W. Payne said that in the subjects of recurrent vomiting the glucose tolerance was normal between attacks, but there was some delay in the storage of lævulose. He had often found the blood-sugar raised at the beginning of attacks. He had noticed that in diabetic children hypoglycæmia was often accompanied by abdominal pain, vomiting, and diarrhœa, which were presumably due to overaction of adrenaline. He wondered whether recurrent attacks of vomiting might be precipitated by a similar mechanism followed by exhaustion of the glycogen reserve of the liver and hypoglycæmia and a further excessive adrenaline action. Dr. Payne, therefore, recommended repeated administration of carbohydrate during the day, between attacks, and a sedative to reduce sympathetic overaction.

Dr. G. M. Kerr said that in practice the best description to use was still "bilious attacks"; he thought that these cases were as numerous as ever, but that fewer were being referred to hospital. The patient might be given a rectal sedative if nothing could be retained by mouth.

The President pointed out that in organic abdominal disease the pain was likely to be more severe, and not so commonly associated with headache and pyrexia. He thought "abdominal migraine" a good descriptive title for these attacks.

Mr. J. T. Edwards described "acetonæmia" in cows, and recurrent vomiting in puppies. Highly selected strains were most liable, and they were also unduly nervous and relatively infertile. Replying to the discussion, Professor Graham said that he had found the blood-sugar to be normal between attacks. He had also seen asthma as a sequel, and he believed this to be commoner than migraine.

## YOUR QUESTIONS

*Correspondents should give their names and addresses (not necessarily for publication) and include all relevant details in the questions which should be typed. Questions of medical interest only will be acceptable for this section. They should be sent in a separate envelope marked "Your Questions". Questions of general interest will be published in preference. Lack of space precludes answering all the questions received.*

**Q. 1.** *Can you suggest any treatment to improve the height of a boy aged 12 years. His height is 4 feet and 2·7 inches. His mental faculties are normal. His father's height is 5 feet and 2 inches and mother's height is 5 feet and 1 inch. His two younger brothers also have a short stature.*

*Is Antuitrin 'S' or testosterone propionate useful for such cases? If so, what is the dosage and how long should the drug be used?*

**Ans. 1.** From the data available it appears that, the retarded height in the child in question is probably hereditary, and genetic in origin. In such cases treatment with hormones is not indicated. Information about the weight, and general development of the child would have been of much help.

However, a thorough examination should be made in order to ascertain the cause of the short stature. This should include a history of the diet and environment, the state of nutrition, determination of the basal metabolic rate, signs of hypogonadism, ratio of the upper and lower skeletal segments, and estimation of the age of the osseous skeleton. At 12 years, the U/L skeletal ratio should be about 0·98. The trochlea, and capitulum at the lower end of the humerus should unite. Hypothyroid dwarfism is attended with infantile skeletal status. In pituitary conditions the skeletal ratio attains proportions of chronological age and epiphysial union is delayed. Hormonal treatment can only be employed when the retarded growth is definitely known to be caused by endocrine disorders. Of all endocrine preparations, thyroid (gr. 1/4) is most useful and effective. Male sex hormone in small doses (methyl testosterone 5 mgm. daily) stimulates growth, especially in presence of hypogonadism. But large doses, and prolonged therapy should be avoided, as these cause inhibition of the pituitary, premature union of epiphyses (therefore retarded growth), and early appearance of secondary sex characters. The pituitary growth hormone available in this country is Antuitrin S (P & D). Pituitary dwarfism is an indication for the use of this drug. The dose required is usually high, 3 to 5 ml. by intramuscular injection daily. The treatment should be occasionally interrupted, for prolonged therapy may cause the formation of anti-hormones in the body. In partial pituitary insufficiency in males, combined therapy with the pituitary growth and male sex hormones gives satisfactory results. The growth effect of hormones is enhanced with vitamins A, D, and $B_1$.

# CURRENT TOPICS

## THE WORLD MEDICAL ASSOCIATION AND THE WORLD HEALTH ORGANIZATION

At the time of the recent meeting of the council of the World Medical Association in Geneva, an informal conference was held between the officials of the World Medical Association and the World Health Organization.

### Medical Education Conference

One of the most important topics discussed was the possibility of holding an international conference on medical education. It was agreed that tentative plans for such a conference would be submitted to the governing bodies of both organizations. The council of the World Medical Association has since voted to sponsor such a conference provided sufficient funds can be obtained. The conference is planned for either 1952 or more probably 1953 and will take place in Europe. The purpose of the conference would be to stimulate improvement of standards of training for medical personnel. Both organizations consider this an essential project, in view of the urgent needs of under-developed regions for additional health personnel.

It was agreed that active co-operation should exist between regional WHO bodies and the World Medical Association. A plan was worked out to facilitate such co-operation.

It was also agreed that there should be fuller co-operation between the two bodies at the planning level. This is to avoid duplication of effort and overlapping of activities.

### Expert Committees of WHO

The matter of representation of WMA on expert committees was thoroughly discussed. It was stated by WHO that no member of an expert committee represented any organization but was chosen only because of his qualifications in the field of activity of the Committee. The World Medical Association is to submit names of experts in various fields to be considered by WHO in establishing panels of experts from which committee members will be selected.

### Naming of Drugs

The matter of the right of the discoverer of a new product or drug to name such item was discussed. WHO reported that sympathetic consideration would be given to the name suggested by such discoverer, provided that the name conformed to generic rules for the naming of such products. The World Medical Association in turn agreed to assist in the acceptance of international non-proprietary names recommended by WHO for drugs and pharmaceutical products, provided due recognition was given to the recommendations of the discoverer of the products.

### Exchange of Information

It was decided that complete exchange of information and documentation should take place between the two organizations.

It was further agreed that contributions would be made by each organization to the publications of the other, with right of each organization to accept or reject such contributions.

WHO agreed to help make known, through one of its publications, the "International Code of Medical Ethics" of the World Medical Association.

### Free Flow of Medical Supplies

The question of free flow of essential medical supplies between countries was discussed. It was made clear that there was no attempt to force on any country the importation of supplies nor to adopt any plan whereby any country might be flooded with useless products. There are, however, in several countries many shortages of supplies, such as DDT, which have resulted from the present world situation. The shortages are in part due to insufficient production and in part due to the lack of transportation. These shortages are crippling some of the activities of WHO in improving health conditions in certain areas.

### Meetings

The advisability of WHO and WMA holding concurrent or consecutive meetings at the same place was thoroughly discussed and did not appear to be feasible. The council of WMA, however, decided that it might be feasible to hold the mid-year session of the council at about the same time as the World Health Assembly and from time to time to hold these meetings in Geneva. This would permit further conferences between WHO and WMA officials and allow members of the council to attend the World Health Assemblies as observers.

Numerous other matters were discussed and taken under advisement for further study by the two bodies.

### Representatives

The World Medical Association was represented in these discussions by: Dr. Elmer L. Henderson, *President;* Dr. Dag Knutson, *President-elect;* Dr. Pierre Glorieux, *Vice-Chairman of the Council;* Dr. Leon Gerin-Lajoie, *Assistant to the Chairman of Council* and Dr. Louis H. Bauer, *Secretary General.* The World Health Organization was represented by: Dr. Brock Chisholm, *Director General;* Dr. Pierre Dorolle, *Deputy Director General;* Dr. S. S. Sokhey, *Assistant Director General* for the Department of Central Technical Services, and various members of the WHO Secretariat concerned with the individual topics under discussion.

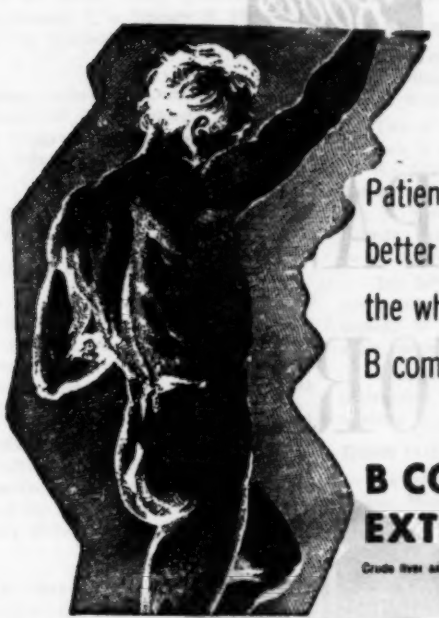
### WHO

The World Health Organization, as a specialized agency of the United Nations, is an intergovernmental body having a total membership of 75 countries and charged with the responsibility, as stipulated in its constitution, to act as the directing and co-ordinating authority on international health work. WHO works with and through the national health administrations of its Member States, and in close co-operation with non-governmental organizations interested in health matters.

### WMA

The World Medical Association, representing national medical associations in 41 countries, is one of these non-governmental organizations. Its objective is to assist in raising levels of health by means of effective liaison among physicians of the world, by the dissemination of medical information, and by the enlistment of co-operation, with other world-wide bodies working in similar fields.

The conference was notable, throughout, for a wholehearted spirit of co-operation, and it is believed that both organizations benefited by the exchange of views. It is hoped that many further such conferences will be held.—*WMA Bulletin,* Vol. 3, No. 3.



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# NOTES AND NEWS

## INDIAN SCIENCE CONGRESS

The 39th session of the Indian Science Congress will be held in Calcutta from January 2 to January 9, 1952, it is reliably learnt. Dr. J. N. Mukherjee will preside over the session.

About 1,600 delegates including a number of foreign scientists as special invitees of the Government of India are expected to attend the Congress. Seventy-one-year-old world famous Physicist Prof. Albert Einstein is expected to attend the session.

Pandit Jawaharlal Nehru, Prime Minister of India, will address the Congress, it is learnt.

## XXVIII ALL-INDIA MEDICAL CONFERENCE

In connection with the ensuring All-India Medical Conference, the Working Committee of the Indian Medical Association will meet on the 13th and 14th February, 1952 at Ahmedabad. The annual meeting of the Central Council of the I.M.A. will be held at Ahmedabad on the 15th and 16th February, 1952.

The All-India Medical Conference will be held on the 17th, 18th and 19th February, 1952 at Ahmedabad. Contributors of scientific papers have to send two copies of the papers to the secretary of the scientific section of the conference at the Prarthana Samaj Hall, Raikhad, Ahmedabad, by the 30th November, 1951.

## XXI PUNJAB PROVINCIAL MEDICAL CONFERENCE, JULLUNDAR

The 21st Punjab Provincial Medical Conference will be held at Jullundar on the 17th and 18th November, 1951. Scientific papers will be read, on the latest advances in Medicine, by some eminent medical men of the State. An Exhibition of medical and allied products is also being arranged for the occasion.

## BOMBAY MEDICAL COUNCIL

The Executive Committee of the Bombay Medical Council has administered a warning to Dr. Frederick Bernard Carvalho, L.R.C.P. (EDIN.), L.R.C.S. (EDIN.), L.F.P.S. (GLASG.), a registered medical practitioner in connection with breach by him of the provisions of section 3 of Part I of the Warning Notice, that he should be more careful in future in issuing his certificates.

## AMENDMENTS TO DRUG RULES

The following amendments in the Bengal Drug Rules, 1946 have been notified:—

I. (1) In the first proviso to rule 3, for the figures 14 in the two places where it occurs, the figure 15 shall be substituted.

II. (1) In schedule C annexed to these rules—(*a*) in item 11 for the word "Penicillin", the words "Penicillin and preparations of Penicillin for parenteral administration" shall be substituted, (*b*) (*i*) after item 11, the following item shall be inserted, namely:—"12. Streptomycin". (*ii*) items 12, 13 and 14 shall be re-numbered as items 13, 14 and 15 respectively.

II. (*2*) In schedule C(1), after item 8, the following item shall be inserted, namely:—"9. Preparations containing Penicillin not in a form to be administered parenterally".

## NUFFIELD FOUNDATION FELLOWSHIPS

The Nuffield Foundation of Britain has offered for India five travelling fellowships for the year 1951-52, the foundation's Liaison Committee in India announced.

Two of these fellowships will be in medical sciences and one each in engineering, natural sciences and social sciences.

An Advisory Committee in India consisting of Sri G. L. Mehta, member of Planning Commission as Chairman, and Sri S. Veradachariar, Dr. C. V. Raman and Mr. J. J. Ghandy, has been appointed by the Foundation to advise it on the administration of the scheme.

The followships, which are normally tenable for one year, are intended for Indian nationals "who have shown unusual capacity to advance knowledge and teaching in one of the fields concerned".

Candidates must be between 25 and 40 years in age, holding preferably a master's or doctor's degree with at least one year's teaching or search experience.—*P.T.I.*

## $50,000 FOR INDIA TO BUY RESEARCH EQUIPMENT

An additional quota of $50,000 has been obtained by the Indian Ministry of Education from UNESCO under the UNESCO international coupon scheme for scientific material, it is learnt. The money will be spent on scientific equipment urgently needed by the research laboratories of the Universities of Calcutta, Madras, Aligarh and Travancore. Other organizations which will benefit are the Indian Institute of Science, Bangalore, the National Metallurgical Laboratory and the Indian Institute of Sugar Technology.

The present quota brings the total received by India since the beginning of this year to $210,000.—*P.T.I.*

## SWISS OFFER TO INDIA

Switzerland has offered India 500,000 francs worth of condensed and powdered milk, tonics and pharmaceutical products for relieving scarcity areas. The relief supplies will be shipped shortly.—*P.T.I.-Reuter.*

## GIFT OF COD LIVER OIL FOR RELIEF OF THE UNDERNOURISHED

A gift of cod liver oil to the value of £500 has been offered by the Indian Famine Relief Appeal of Victoria and accepted on behalf of India by the Minister for Health, the Hon'ble Rajkumari Amrit Kaur, who is arranging to have it distributed among people suffering from undernourishment.

The Indian Famine Relief Appeal grew out of a special meeting called by the Lord Mayor of Melbourne in June, when news of threatened famine in India caused deep concern amongst Australians. The appeal executive has already sent £1,000 to India for the relief of sufferers.—*Australian News Release.*

## FESTIVAL OF BRITAIN

In the Dome of Discovery at the Festival of Britain, Watson and Sons (Electro-Medical) Limited an associate

Company of the General Electric Co. Ltd., have been showing their Cine-Radiography Apparatus. A high power generator and rotating anode X-ray tube in conjunction with a specially designed cine-camera and lens have been used. This apparatus is used for making X-ray cinematograph films of any part of the body. It offers great possibilities in furthering medical knowledge by permitting the leisurely study of the mechanics of moving joints and organs. The equipment was developed in collaboration with Dr. Russell J. Reynolds and was made to the order of the Ministry of Health.

### REFRESHER COURSE IN CARDIOLOGY

Cardiological Society of India has arranged for a refresher course in Cardiology for the general practitioners, to be held three times in the week beginning from the 1st of November next.

The course will consist of twenty lectures by the members of the Society from different parts of India to be delivered at the I.M.A. Hall, at 67, Dharamtollah Street, Calcutta, and four demonstrations in the hospitals.

Admission will be free, on production of admission card, for which and for other details application should be made to the Jt. Hony. Secretary, Cardiological Society of India, 67, Dharamtollah Street, Calcutta 13.

## CORRESPONDENCE

*The Editor is not responsible for any views expressed by the contributors*

### TOXIC MANIFESTATIONS AND THERAPEUTIC FAILURES OF CHLORAMPHENICOL IN THE TREATMENT OF ENTERIC FEVERS

SIR,—In *J. Indian M. A.*, **20**:31, 1950, Drs. Mukherjee and Mitra have published an article on "toxic manifestations and therapeutic failures of Chloramphenicol in the treatment of enteric fever." In this article the writers have stated that with chloramphenicol "toxæmia lessens remarkably" and later on it has been described that, "the drug acted as if it was just an antipyretic without allaying toxæmia." It seems to me that the above-mentioned statements are contradictory; moreover their case reports No. 1 and 3 are in favour of the latter statement that it does not allay toxæmia; my observation on typhoid cases is in agreement with this.

It has been written in the above mentioned article that "parenteral administration of antitoxic sera during the first few days of chloramphenicol therapy has been suggested as a method for neutralising the potent endotoxins from the dead bacilli". As regards the anti-typhoid serum therapy I would like to add a few words. Before the introduction of chloramphenicol in the treatment of typhoid fever I had the opportunity to study the effect of anti-typhoid serum in typhoid cases in the Childrens' Ward of R. G. Kar Medical College Hospitals under Dr. H. Basu, M.B., D.C.H., (LOND.). Let me mention of 3 boys of 10 years. They were moribund cases, 2 boys of 10 years were in typhoid state for about 2-3 days and one female child of 11 years in comatose condition for 24 hours. Toxæmia was markedly diminished at the end of 24 hours after the intramuscular administration of anti-typhoid serum and the patients became conscious between 48 and 72 hours. Each of them survived but the course of the illness was not shortened as it happens with chloramphenicol. These patients were having pyrexia (102°F. to 105°F.) for two weeks. Felix's anti-typhoid serum was not available at that time, so anti-typhoid serum [(natural) B.I.] was administered (50 to 75 c.c. in total).

As I had the impression that chloramphenicol has no effect on toxæmia, I treated two severe cases of typhoid fever with chloramphenicol in conjunction with anti-typhoid serum about 8 months back. They were both male children, one of 13 years, another 15 years; first one came under my observation on the 12th day of continued pyrexia in typhoid state with temperature of 104·5°F., second one on the 16th day of continued pyrexia lying prostrate with circulatory failure, (temperature 101°F.).

Chloramphenicol administered was 5 gms. in divided doses 4 times a day in each case preceded by anti-typhoid serum (B.I.) 100 c.c. injected intramuscularly in two divided doses. Intradermal test with anti-typhoid serum was done before injecting the serum, and lastly injecting anthisan (M & B) 2 c.c. I.M.; all these procedures were conducted to avoid serum reaction. Toxæmia disappeared within 48 hours and defervescence came at the end of the third day; normal diet was given on the 14th day and recovery was without relapse.

I am sorry to point out that the case notes presented in the paper under discussion are so short and incomplete that it is very difficult to compare and contrast the cases mentioned; I shall only refer to few points. Blood pressure was noted in one case only, not in the others, same remarks may be made as regards the hæmatological investigations. As regards the Widal test titres were not noted in figures, whether 'Vi' antibody was titrated could not be understood from it. It has been shown that 'Vi' antibody is of prognostic value. If 'Vi' antibody concentration would have been noted, we would have been in a position to establish some relation as regards the failure or the success of chloramphenicol in low Vi concentration cases because it has been found that low Vi concentration cases run a fatal course.

The authors of the same article have also noted that "in spite of all resuscitative measures patients died of peripheral failure" but we could not have any idea of the "all resuscitative measures" from the article. The peripheral circulatory failure is a very common cause of death in typhoid fever. Of course, this can be prevented as well as combated. In peripheral circulatory failure of typhoid fever we are to combat: (*a*) toxæmia which leads to diminution of contractile tone of terminal arterioles and capillaries with increased permeability of the capillary wall leading to the diminution of circulating blood volume. (*b*) Diminished blood volume with or without superadded acidosis or alkalosis. May I suggest the following as the method of management, which I have tried and have found useful to save the valuable life in the critical period:

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| Vitamin $B_6$ (Pyridoxine) | ... | 1 mg. |
| Nicotinamide | ... | 15 mg. |
| Calcium Pantothenate | ... | 3 mg. |
| Sodium Glycerophosphate | ... | gr. 4 |
| Potassium Glycerophosphate | ... | gr. 4 |
| Calcium Glycerophosphate | ... | gr. 4 |
| Strychnine Glycerophosphate | ... | gr. 1/60 |
| Manganese Glycerophosphate | ... | gr. 1 |
| Lecithin | ... | gr. 1½ |
| Alcohol as preservative | ... | 12% by vol. |

PACKING
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(*a*) first of all blood pressure is to be noted, P. B. Prices' test done every 6 or 12 hours,

(*b*) the foot-end of the bed raised by 1 to 1½ ft.

(*c*) if the temperature is subnormal, body is covered with blanket, hot bags applied. In case of hyperpyrexia, washing of head with cold water or ice bag applied,

(*d*) No hypnotics, even if there is restlessness, or delirium as the hypnotics mask the clinical picture,

(*e*) Glucose 5 per cent alone or with normal saline 500 to 1000 c.c., partly I.V., partly I.M. preferably by continuous drip method to maintain the blood volume. Sodium citrate or acetate is added to it if acidosis is present. Later on 25 per cent glucose 100 c.c. 2 times a day with vitamins C and $B_1$. No fluid per rectum as absorption is indefinite,

(*f*) (*a*) (1) posterior pituitary extract (B.P.) 5 to 10 I.U. atropine sulphate 1/100 gr. 4-hourly or suprarenal cortical extract ½ c.c. to 1 c.c. (*2*) Strychnine sulphate 1/60 gr. 6-hourly or (*b*) Ephedrine hydrochloride ½ gr. 6-hourly. If the patient is comatose without any restlessness and delirium, Methedrin (B.W.) 10 to 30 mgm. I.M. every 6 hours improves the picture.

When the crisis has been overcome anti-typhoid serum is of course to be injected to neutralise the circulating endotoxin. If the patient can take food, sweetened '*ghole*' (butter milk) is given.

The writers of the above article stated that "we suggest proper standardisation and regulation of dosage" of chloramphenicol; everybody will agree with them whole-heartedly because standardisation of dosage will help us to evaluate the role of chloramphenicol in typhoid fever. Vi antibody is to be estimated for the reasons mentioned above. Probably the use of anti-typhoid serum with chloramphenicol will reduce the dosage of chloramphenicol, which is a costly drug. I am using the word 'probably' because my observation is based on only two cases. I am etc.

DEBABRATA PAL.

Ranchi, 26-2-51.

## "ACUTE ABDOMINAL SYMPTOMS WITH PALUDRINE"

SIR,—I shall be highly pleased if you kindly publish the following in the 'Correspondence' column in your Journal.

Dr. N. R. Banerjee, M.B.B.S., D.T.M. of Gorakhpur has published 5 case reports of acute abdominal symptoms with Paludrine in August issue of our Journal (*J. Indian M. A.*, **20**:415, 1951).

Though I always prescribe Paludrine to my patients suffering from malarial fever I never found this complication. I usually advise my patients to take the drug after taking some food *e.g., Moori* or *Khai* (fried rice). So I request my friends to prescribe the drug to the patients with such precaution as taking the drug after some light food. I am etc.

B. K. DEV, M.D., B.S.

The Baroda Clinic,
West Dinajpur, 29-8-51.

# REVIEWS

HOSPITALS—INTEGRATED DESIGN. By Isadore Rosenfield. First edition, reprinted May, 1951. Reinhold Publishing Corporation, 330 W. 42nd Street, New York, N.Y., U.S.A., 398 pages. Prices $15·00.

In the olden days a hospital would perhaps grow up or be set up at places and in fashions that it was just possible to do so, perhaps without any planning at all. But in modern times, and specially during the post war (second world war) period of the last few years, hospital planning has gradually developed into a specialised art and science, and almost a separate entity by itself. Modern hospital planning not only depends on the purse of the promoters, but has to be undertaken with due consideration for numerous other relevant considerations, like purpose of the hospital, site location and lay-out of the surrounding area, the type of the community and density of population it has to serve, problems regarding ventilation including consideration of climate, weather temperature and humidity, water-supply and drainage etc. Mr. Iasdore Rosenfield is a recognised and well-reputed architect who has devoted well-nigh a quarter of a century to this specialised subject of planning and integrated designing of hospitals. His extensive experience has found expression in this wonderful book wherein he has discussed and described various types of hospital designs suitable for almost every type of requirements. The very fact that the first edition of 1947 was sold out, and reprinting had to be taken up as early as 1950, speaks volumes in its favour. Such a book should be on the table of everyone who has anything to do with development, extension, building, design or management of a hospital of any type or requirement.

R. S.

INTERNATIONAL JOURNAL OF SEXOLOGY, Vol. V No. 1 August 1951. Edited by Dr. A. P. Pillay, O.B.E., M.B.B.S. with a board of associate editors from different parts of the world. Annual subscription Rs. 12/- published from Whiteaway Buildings, Bombay-1.

This periodical deals with the delicate matters of sex in an able manner with scientific background. Dr. Pillay has done an immense service in focussing attention to the sexual aspects purely from scientific principles. The issue under review deals with subjects like premature ejaculation, pregnancy in virgins, sodomy, sexual factors in the physician patient relationship, abolition of law of prostitution, divorces, environmental factors and emotional instability, family patterns of social and mental hygiene, frigidity in women by various writers from practically all over globe. The journal contains informations worth investigation by those who want to make themselves familar with sexual behaviour and the many complications that develop in social life. We commend the journal to the medical profession.

# "YOUR HEALTH"

## *Announcement*

Encouraged by the continued assistance and guidance in publishing the Journal of the Indian Medical Association, the Association has decided to publish a new monthly journal YOUR HEALTH from Januuary, 1952. The first issue of YOUR HEALTH is expected to be on sale from the 23rd of December, 1951. The price of each issue will be 12 annas only and the annual subscription will be Rs. 8|- (Post-free); or £0-15s. or $3·00.

YOUR HEALTH will deal with the general rules of healthy living, the various aspects of the prevention of diseases, questions about diet, nutrition, antenatal care, etc.

Health education being in its infancy in India, it is hoped that this health journal published by the Indian Medical Association will help people to gain enormous and useful information pertaining to health. The chief aim of modern scientific medicine and the Indian Medical Association is to assist the people in the attainment of "positive health".

In this venture of the national medical association of India, we solicit the goodwill, advice and whole-hearted co-operation of the members of the profession, the various advertisers of the J.I.M.A. and all others who would be anxious to help us to serve our people in the field of regeneration of their health.

*Details to be had from the office of "Your Health"*
23, SAMAVAYA MANSIONS, CORPORATION PLACE, CALCUTTA-13.

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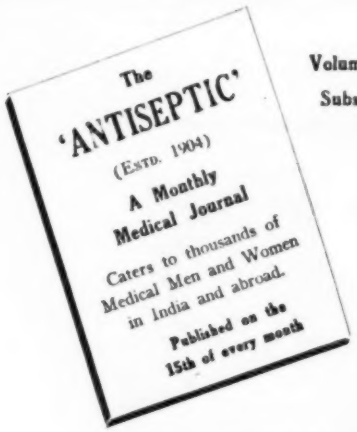


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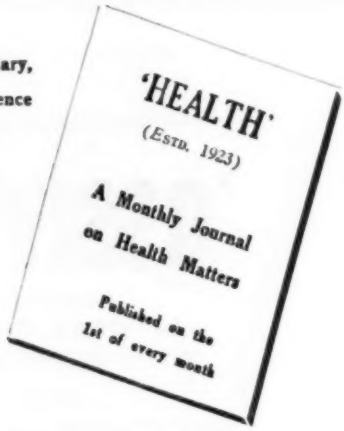


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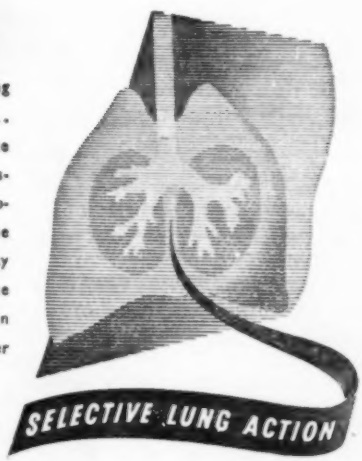


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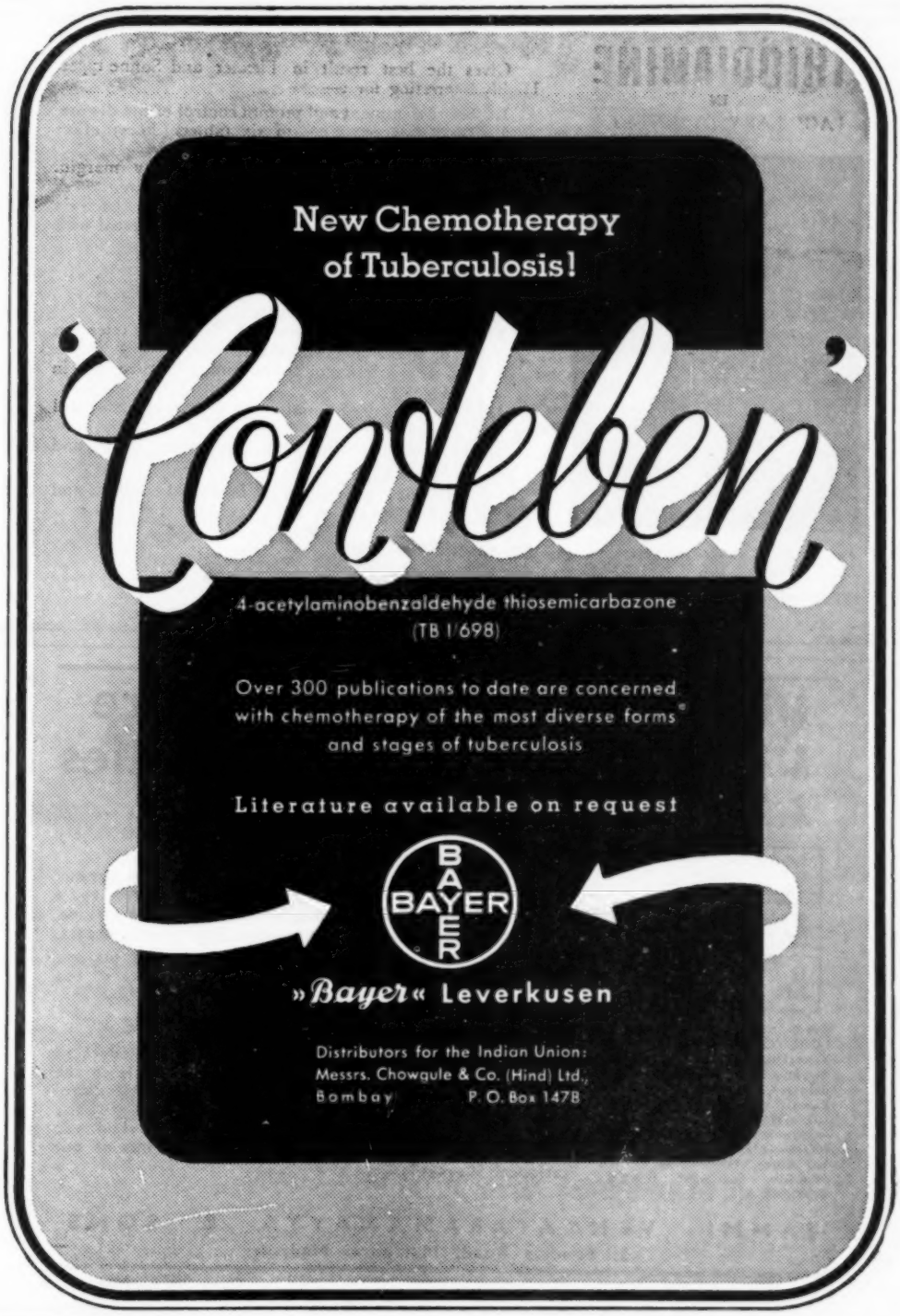
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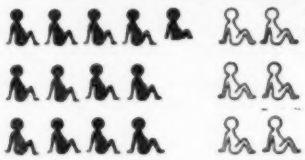

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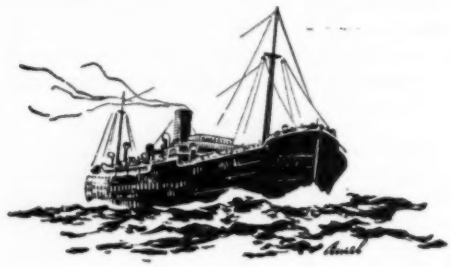

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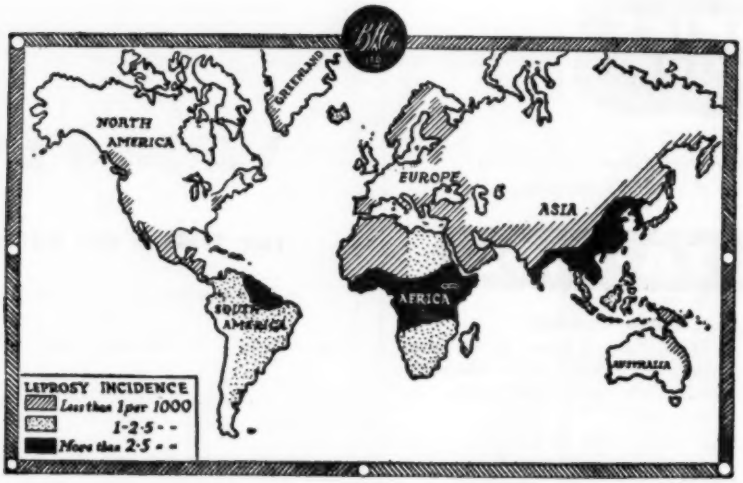


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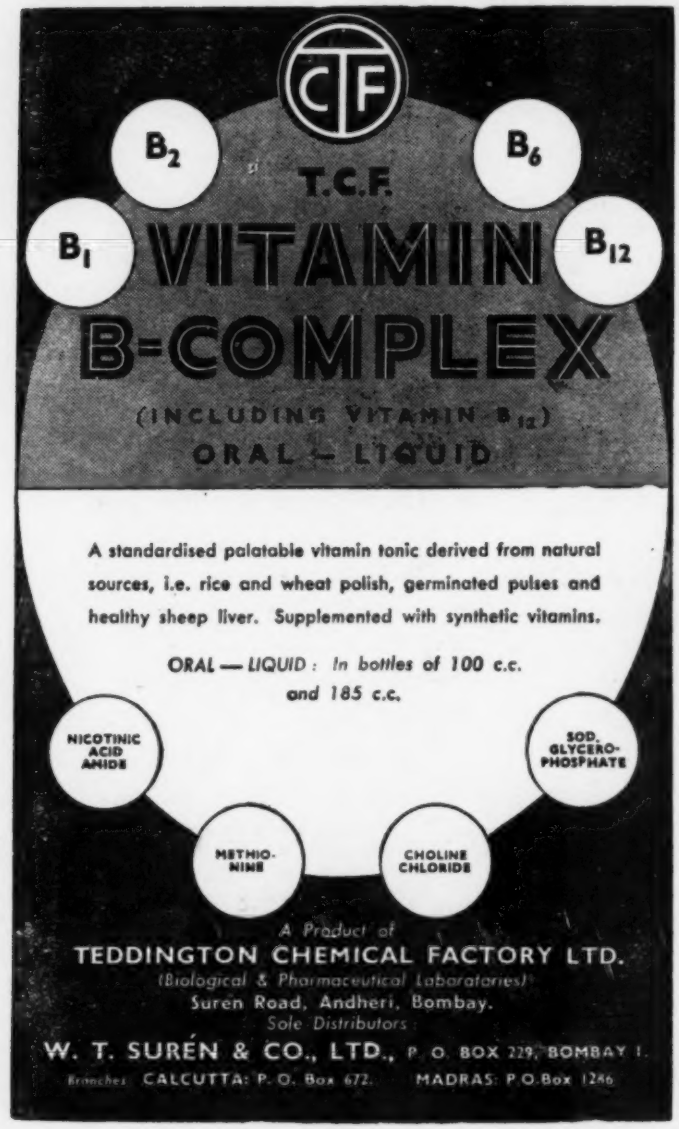
EDITOR—Dr. A. D. MUKHARJI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